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New Orleans Medical
and
Surgical Journal

$4.00 Per Annum, 35c Per Copy
Vol. 104, No. 17

NOVEMBER, 1952

Published Monthly
1430 Tulane Avenue, New Orleans 12, La.

CARDIAC ENLARGEMENT*

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SHREVEPORT

Enlargement of the heart is present in heart disease more often than any other physical finding. An enlarged heart is a diseased heart, or at least a heart working under an added load or strain.

Because of the importance of cardiac enlargement, we felt that it would be of interest and instructive to review a group of enlarged hearts. We have gone over the autopsy material at the Shreveport Charity Hospital from December 1, 1948, to June, 1950, classifying all hearts weighing 350 grams and over as enlarged. We recognize that a 350 gram heart may be within normal limits for a large individual, but generally the normal range is between 250 and 350 grams, or 0.40 to 0.50 per cent of the body weight. The Conference Group on Pathology of the National Research Council gave the range of the normal heart as 275 to 325 grams. There were 200 cases with hearts weighing 350 grams or more in 574 consecutive autopsies, or approximately 35 per cent were enlarged. Table 1 gives the division according to sex and color of the material studied in this series. Our group includes 46 white males, 13 white females, 94 colored males and 47 colored females.

*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, Shreveport, April 30, 1952.

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TABLE 1

1. Number of cases reviewed				574
2. Division according to sex and color:				
White male	116	White female		60
Colored male	240	Colored female		158
3. Number of cases weighing over 350 grams				200
4. Division according to sex and color:				
White male	46	White female		13
Colored male	94	Colored female		47
5. Division according type disease and heart weight				
	350-450	450-550	550-650	650 & up
Hypertensive	76	33	19	8
Syphilitic	5	4	1	1
Coronary disease	18	5	5	1
Calcific aortic dis	2	4	2	1
Myocarditis	..	2	2	..
Miscellaneous	5	4	..	1
Total	106	52	30	12

ETIOLOGICAL CLASSIFICATION

Table 2 gives the etiological classification of the 200 large hearts. As was to be expected, hypertensive cardiovascular disease accounts for the majority of the cases, 136, or 68 per cent. The heart weight in this

TABLE 2
CLASSIFICATION OF HEARTS

			WEIGHT RANGE
1.	Hypertension	136	400-740 gms.
2.	Arteriosclerotic	29	350-700
3	Calcific aortic stenosis	10	400-700
4.	Luetic heart disease	11	360-700
5.	Myocarditis	4	520-630
6.	Bacterial endocarditis	2	400-700
7.	Cor pulmonale	2	400-450
8.	Anemia	3	410-540
9.	Thyrotoxicosis	1	360
10.	Pericarditis (Tbc)	1	520
11.	Congenital	1	500
	TOTAL	200	

group ranged from 400 to 740 grams. Next in frequency was coronary heart disease, or arteriosclerotic heart disease with 29, or 14.5 per cent, the weight ranging from 350 to 700 grams. Calcific aortic stenosis accounted for 10, or 5 per cent, the weight ranging from 400 to 700 grams. Syphilis accounted for 11, or 5.5 per cent. Myocarditis accounted for 4, or 2 per cent, the weight ranging from 520 to 630 grams. The remaining 10 cases, or 5 per cent, were divided between anemia, bacterial endocarditis, cor pulmonale, thyrotoxicosis, pericarditis, and congenital heart disease.

Rheumatic heart disease is actually so designated only once, and that in the group of myocarditis. A large percentage of the cases of calcific aortic stenosis were on the basis of rheumatic heart disease. In the hypertensive group, there were a number of cases with slight to moderate involvement of the mitral valve that were also probably rheumatic in origin. The 2 cases of bacterial endocarditis had aortic valvular disease which may also have been rheumatic in origin. We did not attempt to classify rheumatic valvular disease as a specific group.

The 2 cases of cor pulmonale were in middle aged white males. One had extensive pulmonary tuberculosis and fibrosis, the other had diffuse lymphatic carcinomatosis of the lungs secondary to a small gastric cancer. The thyrotoxic patient was a young colored female who died in congestive failure from thyrotoxic heart disease. The tuberculous pericarditis was in an 18 year old colored male who also had a considerable degree of myocarditis. He, too, died in congestive failure. The congenital heart in this group was that of a 12 year old white female who had arachnodactylia, a dilated aortic valvular ring and a moderate coarctation. She died suddenly, presumably from a cardiac arrhythmia.

The hypertensive group has been further classified as in Table 3. Of the 136 cases in this group, 83 or approximately 61 per cent were classified as essential hypertension. There were 15 cases of malignant hypertension, approximately 11 per cent of the group. Their ages varied from 29 to 68 years, 6 of them falling between 40 and 50 years. All were in colored patients, 9 males and 6 females. The heart varied from 400 to 740 grams, and there were 8 cases with hearts weighing 600 grams or more. The enlargement was predominantly of the left ventricle.

TABLE 3
HYPERTENSIVE HEARTS

		W. M.	W. F.	C. M.	C. F.
1.	Entire group	46	13	94	47
2.	Hypertensive	27	7	68	34
			Age Range	Weight Range	
3.	Malignant Hypertension	15	29-68	400-740 gms.	
4.	Renal Hypertension				
	a. Pyelonephritis or pyonephrosis	30	41-78	400-620	
	b. Polycystic Kidney	3	46-77	460-520	
	c. Intracapillary Glomerulosclerosis	4	32-67	360-440	
	d. Periarteritis Nodosa	1	32	650	
5.	Essential Hypertension	83			
	TOTAL	136			

There were 38 cases, or 28 per cent, in the hypertensive group that were classified as renal hypertension. Of these, 30 were due to pyelonephritis or pyonephrosis. Their ages varied from 41 to 78 years, and the heart weight was from 400 to 620 grams.

There were 3 cases of polycystic kidney disease. Their ages were 46, 74, and 77 years, and the heart weight 400, 520, and 520 grams. There were 4 cases of intracapillary glomerulosclerosis, or Kimmelsteil-Wilson disease, varying in age from 32 to 67 years. The heart weight varied from 360 to 440 grams. One patient had periarteritis nodosa, a late development in his hypertensive disease. He was 32 years of age and the heart weighed 650 grams.

CAUSE OF DEATH

Table 4 lists the cause of death in the hypertensive group. In the malignant hypertensive group 11 died from uremia and 4 from cerebrovascular accidents. In the renal and essential hypertension group, 26 died from cerebrovascular accidents and 16 from uremia. Twenty died in congestive

TABLE 4
CAUSE OF DEATH IN THE HYPERTENSIVE GROUP

1.	Malignant Hypertension	15
	a. Uremia	11
	b. C. V. A.	4
2.	Renal and Essential Hypertension	121
	a. Uremia	16
	b. C. V. A.	26
	c. Congestive Failure	20
	d. Sudden Cardiac Death	7
	e. Pulmonary Emboli	7
	f. Pneumonia	9
	g. Miscellaneous	36
	TOTAL	136

failure, and there were 7 sudden deaths, probably due to cardiac arrhythmia.

It is of interest to note the distribution of the entire hypertensive group according to color. Seventy-two per cent of the enlarged hearts in the colored group were due to hypertension, while only 57 per cent of the hearts in the white group were due to hypertension. The higher incidence of hypertensive disease in the colored race has been noted in the hospital population for many years.

There were 29 cases of coronary artery disease, which are further subdivided in Table 5. There were 12 cases of severe coronary sclerosis, and 11 coronary artery occlusions with myocardial infarction. It is to be noted that there were 4 myocardial infarctions without coronary occlusion, on the basis of myocardial anoxia. They all had marked coronary sclerosis and large hearts, 450, 530, 550 and 550 grams each. Three also had hypertension. One died from cerebral hemorrhage, 1 during an operation, 1 from postoperative pulmonary embolus, and 1 from congestive failure. There were 2 coronary occlusions without infarctions, both in white men, age 65 and 82 years. Probably they had developed over the years a good collateral circulation. (Table 6) One had a complete occlusion of the right coronary about 2 cm. from its orifice, and the other an occlusion of the left anterior descending coronary. One died suddenly. The other also had purulent pericarditis and died from toxemia and congestive failure.

It is also interesting to note that 23 or 79 per cent of this group were over 60 years

TABLE 5
CORONARY ARTERY DISEASE

			Weight	Age
1.	Severe coronary sclerosis	12	350-440 Gm.	42-82
2.	Coronary occlusion with infarct	11	370-700	36-78
3.	Infarction without occlusion	4	450-550	40-80
4.	Cor. occlusion without infarct	2	380-400	65-82

Percentage of the Entire Group Having Coronary Artery Disease

1. 18 of the 46 white males or 39%
2. 4 of the 13 white females or 30.8%
3. 3 of the 94 colored males or 3.2%
4. 4 of the 47 colored females or 8.5%

Percentage of the Total White and Colored With Infarction

1. 13 of the 59 white males and females or 22%
2. 2 of the 141 colored males and females or 1.4%

Age Groups

30 to 40	1
41 to 50	3
51 to 60	2
61 to 70	14
71 to 80	5
81 & up	4
TOTAL	29

TABLE 6
CAUSE OF DEATH IN CASES OF CORONARY ARTERY DISEASE

Coronary Occlusion and Infarction

1.	Sudden death	6
2.	Shock	4
3.	Congestive failure	4
4.	Pulmonary embolus	1
5.	Miliary tuberculosis	1
6.	Cerebral hemorrhage	1
	Total	17

Coronary Sclerosis

1.	Sudden death	2
2.	Congestive failure	1
3.	Pulmonary embolus	1
4.	Subarach. hemorrhage	1
5.	Miliary tuberculosis	1
6.	Pneumonia	2
7.	Pyelonephritis	2
8.	Carcinoma	1
9.	Ruptured aneurysm (cardiac tamponade)	1
	Total	12

of age. Of the 15 with infarcts there were 10 or 66.7 per cent over 60 years of age. In the entire group there were 22 or 76 per cent in the white race, and 7 or 24 per cent in the colored race. When they are divided according to color and sex, we find that 18 of the 46 white males, or 39 per cent, and 4 of the 13 white females, or 30.8 per cent, had coronary artery disease as compared to 3 of 94 colored males or 3.2 per cent, and 4 of 47 colored females or 8.5 per cent. In the myocardial infarction group there was only 1 colored male and 1 colored female. The preponderance of coronary occlusions and myocardial infarction in the white race in the hospital population has been repeatedly noted on the wards and at autopsy. There were 13 of the 59 white patients who had myocardial infarction or 22 per cent as compared to 2 of the 141 colored patients, or 1.4 per cent. Table 6 gives the cause of death in those with coronary sclerosis and those with infarction.

There were 10 cases of marked calcific aortic stenosis. (Table 7) Seven were in colored males, 2 in white males, and 1 white female. In 5 cases hypertension was present and 9 cases had varying degrees of coronary sclerosis. Five of the group died from congestive heart failure. The 1 white female was 49 years of age, 1 colored male was 52 and 1 was 56 years of age. The remainder were all 65 years and older, one being 95 years of age with a heart weighing 500 grams. This group illustrates the fact that cardiac hypertrophy is compatible with good function for years. Table 8 lists the 11 syphilitic hearts.

TABLE 7
CALCIFIC AORTIC STENOSIS

1.	Number of cases	10
	Colored male—7, White male—2, White female—1.	
2.	Age range 49 to 95, Six over 60 years old.	
3.	Heart Weight—400 to 700 Gms., Six over 500 Gms.	
4.	Cause of death: Congestive failure	5
	Pneumonia	2
	C. V. A.	3
5.	Number with associated hypertension	5
6.	Number with associated coronary sclerosis	9
	Moderate—6, Severe—3	

TABLE 8
SYPHILITIC HEART DISEASE

Syphilitic aortitis and valvulitis	11
Associated aneurysms	4
Narrowing of coronary ostia	4
Coronary arteriosclerosis	5
Associated hypertension	1
Weight range	360-700 grams
CAUSE OF DEATH	
Congestive failure	7
C. V. A.	1
Tuberculosis	1
Ruptured aneurysm	1
Accidental	1

Table 9 lists the 4 cases of myocarditis, 1 rheumatic without valvular disease, and 3 of Fiedler's myocarditis. They all had very large hearts and died in congestive failure. One of the Fiedler's myocarditis cases was a colored male who had had heart disease for several years, thought to be rheumatic, but at autopsy proved to be Fiedler's. The other 2 Fiedler's were in young colored women, 21 and 24 years. One was definitely postpartal and the other probably so. Every heart weighed over 500 grams, showing both hypertrophy and dilatation. This group offers a striking contrast to the preceding group with calcific aortic stenosis. They all had large hearts, were relatively young, and were in congestive failure. The hypertrophy and dilatation were due to impaired functional capacity of the myocardium. Congestive failure occurred early in the course of the disease, and was persistent.

There were 2 cases due to sickle cell

TABLE 9

	MYOCARDITIS		
1.	Rheumatic myocarditis	1	Colored Male
	Heart weight 600 gms. Valves normal		
	Died in acute congestive failure		
2.	Fiedler's myocarditis	3	1 CM, 2 CF
	Weights—520, 540, 600 grams		
	Expired suddenly	3	
	ANEMIA		
1.	Sickle cell	2	1 CM, 1 CF
			Age 18 Age 46
	Heart weight		540 410 gms.
	Cause of death: Sickle Cell Crisis		2
2.	Pernicious Anemia	1	WM age 77
	Weight		420 grams
	Cause of Death: Congestive failure & pneumonia		

anemia, 1 colored female 46 years of age, and 1 colored male 18 years of age. The hearts weighed 540 and 410 grams. The myocardium was described as reddish brown color without scarring. The coronary arteries were patent in both cases; there was a mural thrombus in the right auricular appendage in one with pulmonary infarction. They both died in sickle cell crises, one about nine hours and the other about forty-eight hours after admission.

The cardiac involvement in sickle cell anemia, according to Margolis[6] can be explained on the basis of four factors, or a combination of them: (1) Chronic anoxia of the myocardium because of severe anemia of long duration. (2) Circulatory stasis, producing thrombosis of the small and medium sized arteries of the lungs, which progress to an organized state. This may increase the pressure in the pulmonary arteries and lead to right ventricular enlargement (cor pulmonale) and right heart failure. (3) Circulatory stasis producing thrombosis of the coronary arterioles, resulting in damage and consequent scarring of the myocardium. (4) The severe anemia which produces a chronic anoxia of the body tissues and increases the demand for more oxygen, and thus leads to an increase in cardiac output, which may cause the myocardium to hypertrophy and dilate.

There was 1 case of pernicious anemia in a white man 77 years of age, whose red cell count was 3,160,000 with 9 grams of hemoglobin on admission. The heart showed moderate hypertrophy, weighing 420 grams. The left ventricular wall measured 1.5 cm. and the right 0.5 cm. He also had pneumonia, chronic active pyelonephritis, early lower nephron nephrosis, and chronic passive congestion of the liver.

All of the aneurysms have been grouped in Table 10. There were 4 luetic and 8 arteriosclerotic. Of the arteriosclerotic there were 4 dissecting and 4 fusiform. Of the 4 dissecting aneurysms, 2 were patent and the patients died of cerebral hemorrhage. The other 2 died from rupture of the aneurysm, 1 into the pericardium with cardiac tamponade, and 1 into the left pleural cavity. Four of the group died from congestive failure.

TABLE 10
ANEURYSMS

1. Luetic	4	Ruptured	1
2. Arteriosclerotic—			
a. Dissecting	4	Ruptured	2
		Patent	2
b. Fusiform	4	Ruptured	1

WEIGHT RANGE

Luetic—360-370-450-500 grams
Dissecting—400-440-500-700 grams
Fusiform—400-420-450-700 grams

CAUSE OF DEATH

Ruptured aneurysm	4
Congestive failure	4
Cerebral hemorrhage	2
Pneumonia	1
Tuberculosis	1

In Table 11 we have listed all of the hearts weighing 550 grams and over. There were 42 in the group, 27 of which were due to hypertensive disease, 6 to coronary artery disease, and 4 to calcific aortic stenosis. There were two of the cases of myocarditis with hearts weighing over 550 grams. Of this group of 42 very large hearts, 15 died from congestive heart failure, and 6 died sudden cardiac deaths. Of the 9 cases dying in uremia, 8 were in the malignant hypertensive group.

TABLE 11
HEARTS WEIGHING 550 GRAMS AND OVER

1. Hypertensive	27
2. Coronary artery disease	6
3. Calcific aortic stenosis	4
4. Bacterial endocarditis with aortic valvular disease	1
5. Syphilitic heart disease	2
6. Myocarditis	2
TOTAL	42

CAUSE OF DEATH IN THIS GROUP

1. Congestive failure	15
2. Sudden cardiac death	6
3. Cerebro-vascular accident	5
4. Pulmonary emboli	3
5. Uremia	9
6. Miscellaneous	4

CARDIAC COMPENSATORY MECHANISMS

There are 3 cardiac compensatory mechanisms: acceleration of the beat, dilatation, and hypertrophy. The acceleration of the beat, or tachycardia, is usually a transient compensatory mechanism. Dilatation and hypertrophy, which augment the total

output by increasing the output per beat, provide a more efficient and more permanent means of compensation.

Cardiac enlargement is the result of a combination of dilatation and hypertrophy. Usually, dilatation precedes hypertrophy and in very large hearts accounts for the major increase in size. It actually denotes an increase in the capacity of the heart chambers and is the commonest manifestation of any cardiovascular disease which tends to impair the cardiac output.

Cardiac dilatation, according to Freidberg,[2] results from: (1) mechanical lesions which tend to increase the diastolic volume and thereby cause cardiac dilatation by (a) increasing the venous return to the heart, e.g., in cases of arteriovenous aneurysm; (b) increasing the resistance to output from one of its chambers, e.g., mitral or aortic valve stenosis or hypertension; (c) permitting a reflux of blood into chamber after it is ejected, e.g., in aortic valvular insufficiency; (2) myocardial lesions which tend to impair cardiac contraction and diminish cardiac output while the inflow is temporarily unchanged. This causes an increase in the diastolic volume and cardiac dilatation, e.g., myocardial infarcts secondary to coronary occlusion, and rheumatic myocarditis.

Cardiac dilatation is a compensatory mechanism in accordance with Starling's Law of the Heart: "Within physiologic limits the larger the volume of the heart, the greater is the energy of its contraction, and the amount of chemical change at each contraction." It is largely specific in that the entire heart is not involved, but chiefly that side or chamber upon which the load has been increased.

The third compensatory mechanism of the heart is hypertrophy, which is usually preceded by dilatation. It is also selective in that it develops in the chamber or chambers of the heart that have been placed under an added load or strain. Hypertrophy results from increased work, or as stated by Fishberg,[1] the mass of the myocardium is a function of the work it performs, with the limitation that the work must be considered in relation to the functional fitness of the myocardium. The increase in mass, or hypertrophy, results when the work of a cardiac chamber is increased in an absolute sense, such as one sees in hypertension and valvular defects. Secondly, hypertrophy may develop when the functional capacity of the myocardium is impaired, even though the work it is called upon to perform is not elevated. There results a relative increase in work, in that the work performed approaches more closely to the maximum of which the heart is capable. Hypertrophy of this nature is seen in the various forms of myocarditis, rheumatic or nonspecific, and in degenerative lesions resulting from coronary arteriosclerosis.

Fishberg[1] states that hypertrophy follows: (1) In health, when as a result of physical exertion the work of the heart is increased. (2) When the resistance to the expulsion of the blood is increased by valvular stenosis or hypertension. (3) When in addition to expelling the blood, the heart has to drag the chest wall with it during systole, as occurs in some cases of adhesive mediastinopericarditis. (4) When the volume of blood expelled per stroke is increased, as in valvular insufficiency, and some instances of Grave's disease and anemia. (5) When the functional capacity of the myocardium is decreased for a considerable time, as a result of inflammatory or degenerative lesions.

Hypertrophy is accomplished by an actual increase in muscle mass, due to an increase in the thickness of the individual muscle fibers, and not from an increase in number of muscle fibers. Normally, the average diameter of the muscle fiber is between 15 and 20 microns. In the hypertrophied heart, it is generally above 25 or 28 microns. With increase in size of the individual muscle fibers, there is not an increase in the number of capillaries, and as a result, an actual decrease in concentration of capillaries results. Therefore, each capillary is called upon to supply a larger mass of muscle, and this mass increases proportionally with the weight of the heart. As the thickness of the individual muscle

fibers is increased, it requires a longer time for completion of the metabolic exchanges with blood. This decrease in capillary muscle ratio is certainly one of the main factors responsible for the inefficiency of the large hearts.

Cardiac hypertrophy is a compensatory mechanism which permits the heart to maintain a normal circulation despite a cardiovascular lesion and also to retain an adequate reserve for unusual circulatory demands. This has been frequently illustrated in this series by the number of large hearts resulting from hypertension and calcific aortic disease in individuals who have lived to be 60 or more years of age. In other words, hypertrophy without myocarditis or degenerative changes from coronary disease is compatible with years of normal activity.

Cardiac dilatation and hypertrophy are reversible. It has been frequently noted that cardiac enlargement from dilatation and hypertrophy has regressed with removal or correction of the cause. Such has been observed (1) after surgical correction of an arteriovenous aneurysm, patent ductus arteriosus, coarctation of the aorta, and hyperthyroidism; (2) sympathectomy in hypertensive heart disease; (3) correction of anemia; (4) adequate thyroid administration in myxedema; (5) vitamin B in beriberi heart disease.

It is important that we diligently search for cardiac enlargement and more specifically for the individual chamber or chambers that are enlarged. The accuracy of our diagnosis rests in a large degree upon this examination. We have at our disposal three common methods of examination: physical examination, x-ray examination, and the electrocardiogram. The first of these is the least spectacular, but is always with us, requires no special apparatus, and can be done at home or in the hospital. A careful physical examination for determination of heart size should be assiduously practiced. It should not become a lost art.

SUMMARY

1. We have presented a statistical study of 200 cases of cardiac enlargement occurring in 574 consecutive autopsies at the Shreveport Charity Hospital.

2. The percentage of cases with hypertensive disease in the colored race is significantly higher than in the white race, while the percentage of cases with coronary artery disease in the white race is far greater than in the colored.

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PAROXYSMAL TACHYCARDIA*

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SHREVEPORT

The term paroxysmal tachycardia embraces that group of so called cardiac arrhythmias characterized by excessively high rate, regular rhythm, usually with a sudden onset, and frequently, with an abrupt return to normal. In the broad sense it includes auricular fibrillation and flutter, and ventricular fibrillation, but these are excluded from this discussion. The condition results from a succession of premature or ectopic beats arising from a focus in the auricular, A-V nodal, or ventricular tissue. The point of origin of the extra systoles determines the type of tachycardia, and,

*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, April 30, 1952, Shreveport, Louisiana.

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with few exceptions, it is necessary to rely on the electrocardiogram to establish the type. Most writers now differentiate only between ventricular and supraventricular types of tachycardia. This is the only basis on which there are differences in the etiology, clinical picture, and treatment of the condition.

Paroxysmal tachycardia is said to be a rare condition: Leaman reports only 17 instances in 3000 consecutive electrocardiograms. Perhaps it is the difficulty, and even anxiety, experienced by the writer in several recent cases which creates for me the illusion of frequency. These factors have undoubtedly led me away from the widely held feeling that paroxysmal tachycardia is a simple, innocuous disturbance which requires little or no treatment. It is the desire to share these experiences with you, which prompted the selection of this title.

CASE REPORTS

The youngest of our patients was only eight days of age when his tachycardia developed. He was a well formed infant of normal weight and was delivered normally. There was no evidence of cardiac disease or anomaly. Tachycardia was supraventricular in type with a rate in excess of 200. Reversion to normal sinus mechanism followed administration of digitoxin. Tachycardia recurred but subsided after the dose of digitalis was increased. The process covered a period of about twenty-four hours.

An emergency call late one afternoon revealed a prominent business executive, 48 years of age in acute distress with tachycardia of only ten minutes duration. He was in good general health, and had no organic heart disease. There had been one previous episode of tachycardia about three years ago. Prior to this attack he had been working strenuously and long, and much of the overtime work had been in the nature of entertaining visiting business associates. The patient was prostrate and pale, the lips and nails cyanotic. The heart rate was about 180 per minute and the blood pressure measured 84/80. Carotid pressure produced no appreciable change in cardiac rate, nor did left ocular pressure. Just as the attempt was about to be abandoned, (because of inability of the patient to tolerate the severe pain), pressure on the right eye resulted in a sudden drop in heart rate to normal and rise in blood pressure to 130/80. These changes were associated with complete clearing of all symptoms and signs of cardiovascular disturbance. A period of hospitalization followed, and all examinations revealed normal findings.

It was at first thought that this might be a case of ventricular tachycardia but the fact that ocular pressure brought relief classifies it as supraventricular in type. It affords evidence that supraventricular tachycardia may be a serious condition, for with a pulse pressure of only 4 millimeters of mercury there is, obviously, a marked deficiency in circulation. The possible complications and sequelae in an episode of this type are much more formidable than with those in which the blood pressure remains at a reasonable level, but even under such conditions myocardial injury may occur. The case of a young adult is recalled wherein supraventricular tachycardia persisted for more than forty-eight hours. There was little discomfort and little disturbance of circulation but the electrocardiogram retained inverted T-waves for more than a week after normal mechanism was reestablished.

A fatal outcome was barely avoided in the case of Mr. C. G. H., a 52 year old automobile salesman. He came to the hospital at 2:30 A. M., on February 11, 1952, and had to be assisted into the examining room. He had been awakened from sleep shortly after 10 P. M. by severe epigastric pain and nausea. He had vomited several times. Suspecting the presence of a surgical condition he had called a surgeon, who promptly asked for medical consultation. Morphine and atropine had been given with some degree of relief of the epigastric pain and oxygen therapy had been started. The patient was ashy in color, with profuse perspiration and the temperature was 97° F. There was no palpable pulse and no measurable blood pressure. The cardiac rate was very rapid (220 per minute) and perfectly regular. Carotid pressure produced no change in the picture. Ocular pressure caused a temporary minimal degree of slowing of the heart rate. An electrocardiogram was made then, and revealed ventricular tachycardia. Pronestyl was administered intravenously in glucose, and after 250 mgms., it was discontinued when slight slowing of the heart rate and alteration of the electrocardiographic pattern was noted. This effect was very transient, however, and twenty minutes later another dose of 250 mgms. was given with practically no result. By this time, 3:45 A. M., the patient had become comfortable but there was still no palpable pulse or audible blood pressure. He was moved to his hospital room, oxygen continued, and was given 3 grains of

quinidine. Following the slight exertion of the move the cardiac rate returned to its original extremely high level. At 5:00 A. M. a 500 mgm. dose of pronestyl was given. The rate dropped to 160 per minute and the radial pulse became perceptible with every second or third systole. Improvement in the color of the skin was noted also, and it was felt that progress was being made. Quinidine was continued, 3 grains every two hours until five doses were given. It was then after noon and the tachycardia had again resumed its rate of 220 per minute. The condition of the patient was unchanged except that he was fairly well under barbiturate sedation and was not apprehensive. At 4:00 P. M. another injection of 500 mgs. of pronestyl was given with no response. After the passing of another hour it was felt that something had to be done, and despite the fact that it is usually of no avail in such cases digitalis was given. This was administered in the form of digitoxin, 0.8 mgm. in glucose, intravenously. This resulted in no immediate slowing of the rate. No further medication was given that evening except sodium luminal. At 11:00 P. M. the pulse was found to be 74 per minute, and the blood pressure 142/90. Digoxin 0.25 mgm. was given twice on the following day, then discontinued. On this date, February 12, there was a loud pericardial friction rub beneath the sternum. It persisted throughout the day but was not heard at any later time. During this day, there was troublesome hiccough but this disappeared with the friction rub. Mild precordial pain was noted at intervals through February 15, but after this there were no symptoms or complications.

An electrocardiogram on February 13, showed inversion of T waves in the conventional leads and in V-6. T. waves in lead 1 and V-6 have since become upright, but the findings of posterior infarction have persisted. During the acute phase the leukocyte count rose to 17,000 with 80 per cent neutrophiles, but there was no febrile rise above 99° F.

Patients who present the Wolff-Parkinson-White syndrome of short P-R interval with wide QRS complex are particularly subject to attacks of paroxysmal tachycardia. In most instances the episodes are supraventricular in type, a few are of ventricular origin, but it is rare to find an individual who at different times presents both types of tachycardia. This is illustrated by the following case.

Miss L. L. was eleven years of age when seen in August 1949, with her first attack of rapid heart action. She was in good general health and had always led an active life. Examination was normal except for the heart rate which was counted to be approximately 280 per minute. Carotid pressure did not slow the rate. As the patient was not in distress, she was given sodium luminal and urged to sleep. Spontaneous emesis after a short while resulted in normal heart action. An electrocardiogram was not obtained during this episode, but one made several days later revealed the findings of the Wolff-Parkinson-White syndrome.

A second attack of tachycardia occurred in January 1950. With this, there was pallor, cyanosis and shock. The rate was 240 and the blood pressure was 88/80. Quinidine and phenobarbital were given by mouth but nausea and vomiting forced temporary withdrawal of these drugs. Digitoxin was given parenterally, then quinidine used again with dosage reduced to 1-1/2 grains every two hours. The duration of this attack was twenty-six hours. During and following this episode there were fever and chest findings which were the result of atypical pneumonia or pulmonary thrombosis.

On March 7, 1950, the child was brought to the hospital from school, again having tachycardia. She was tense, anxious, complained of tightness in the chest, and nausea was present. Emesis followed each oral dose of quinidine. Sodium luminal was given by needle until sedation was evident and anxiety disappeared. The electrocardiogram had demonstrated ventricular tachycardia with a rate of 240. Quinidine for intravenous administration was being prepared, when the heart rate dropped to 160, so its use was delayed. About two hours later normal findings were present. Quinidine was then given by mouth, was well tolerated and was continued for several days.

In June 1950, tachycardia developed while swimming. There were few subjective symptoms, and no anxiety. This was of supraventricular type with a rate of 180 per minute. Rest and increased doses of quinidine and phenobarbital resulted in cessation of tachycardia after a period of about two hours.

No further difficulty was experienced until February 22nd, 1951. While at school just before lunch a few short runs of extrasystoles were noted. The meal was eaten, but not heartily, and shortly thereafter tachycardia ensued. At the clinic it was found that this was ventricular tachycardia with the rate again 240 and the blood presure 80/70. She was pale, the skin moist and the pupils dilated. She was given 2 grains of sodium luminal by needle, then 200 mgms. of pronestyl by intravenous injection. Within 90 seconds after receiving this medication the rate had dropped to 120 per minute and there had been a sudden transition to the normal mechanism.

One of the first holiday dances in December 1951, was interrupted for this young lady, now 13 years of age, by the onset of another episode of ventricular tachycardia. The clinical picture was

the same as on previous occasions, with mild shock, blood pressure 80/70 and the rate 250 per minute. Two injections of pronestyl were given within thirty minutes with the total dose of 500 mgms. Each of the injections produced definite slowing of the rate with alteration of the electrocardiogram, but an additional 50 mgms. per hour for three doses was needed to produce complete control of the tachycardia. There has been no recurrence, but on two occasions pronestyl has been taken by mouth when the presence of extra systoles has been noted. It is anticipated that elimination of the factors of adolescence, increased emotional stability, and the ability to recognize premonitory signs may all be helpful in avoiding these disabling seizures in the future.

The fact that supraventricular tachycardia is usually found in individuals with hearts which are organically sound does not preclude the possibility of its association with cardiac disease. This is demonstrated by Mr. F. S. who is sixty-three years of age with quite marked generalized arteriosclerosis. Although he had never had tachycardia prior to eighteen months ago, he is at present hospitalized for the sixth time since June 1951, for treatment of supraventricular tachycardia or because of myocardial failure following such an episode. With the added appearance of anginal pain even while at rest the outlook is far from bright.

TREATMENT

This discussion has presented only the gloomy side of the picture, but it is a fact that the sun is brighter on the upper side of the cloud. From that vantage point we see that ventricular tachycardia is infrequently encountered and that the supraventricular episodes are usually easily controlled. With the latter group success will result more often than failure if carotid sinus pressure is applied properly to first one side and, if necessary, to the other. Ocular pressure will produce reversion to normal mechanism in a fair percentage of the cases not relieved by carotid stimulation. It should be pointed out that this maneuver calls for real pressure on the eye ball, and that no result may be expected unless this is carried to the point of marked discomfort to the patient. Ocular pressure can be applied best with the thumb, carotid pressure with the fingers, while standing on the opposite side of the patient and reaching across. In either case, the patient should be supine, and my preference is for the head to be in the midline. Neither of these procedures is carried out by the writer without the stethoscope on the precordium of the patient and pressure is released with the very first suggestion of change in mechanism. Even so the period of cardiac inactivity during reversion often "sounds" long, and the first normal systole has a beautiful tone.

For those cases, which do not respond to the above procedures, many things have been advocated. Induced vomiting is the most simple, but except in the first episode of tachycardia, will probably have been tried before the patient seeks medical aid. Mecholyl will undoubtedly control most of these attacks but the severe side effects and the danger attendant to its use have dissuaded me from its employment. In the instances in which intravenous neo-synephrine might have been used there have been other factors in my cases which seemed to constitute contraindications. Quinidine is the drug of choice, but the dosage must be adequate. If this fails digitalis should be employed, and always if there is associated myocardial failure. Digitalization may need to be complete and urgency may indicate the intravenous route using either cedilanid or one of the new glycosides. It is fortunate that in the majority of instances the general condition of the patient is such that great haste is not necessary, and with these individuals the more simple of the above measures together with sedation, rest, and time will result in reversion before there is any real damage.

CONCLUSION

In conclusion it may be stated again that while most cases of paroxysmal tachycardia are relatively benign and easily controlled affairs, there are those of both ventricular and supraventricular type which may produce many anxious moments for both patient and physician. Several cases of the latter group have been reported, and general measures in the treatment of these conditions have been discussed.

THE DIAGNOSIS OF CORONARY PAIN*

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NEW ORLEANS

One of the most common and at the same time one of the most difficult problems which the physician encounters is that represented by the patient whose symptoms suggest that coronary insufficiency may be present. The complaints are often such that a variety of conditions must be considered such as neurosis, diaphragmatic hernia, cervical arthritis, mediastinal disease, pericarditis, gallbladder disease, and diseases of various other upper abdominal organs. The essential difficulty, however, resides in the fact that it is so often not easy to know whether coronary disease is present or not.

DIAGNOSIS

In our attempt to learn whether or not a patient's discomfort is due to coronary disease we employ three methods:

1. History
2. Electrocardiographic studies
3. Response to nitroglycerine.

When the patient's symptoms follow one of the typical descriptions found in the literature there is seldom much doubt about the diagnosis. If, in addition, there is a prompt response to nitroglycerine there is no doubt about the diagnosis. Unfortunately, in a great number of cases the symptoms do not follow the description furnished in the literature, and it is with these cases that we have so much difficulty.

The electrocardiogram has furnished one means by which these cases can be investigated, but it must be used properly if we are not to be misled. In most instances the electrocardiogram is perfectly normal between attacks. Therefore, attempts must be made to make electrocardiograms while the attack is present. In a patient whose attacks are not frequent this may involve considerable difficulty. It involves racing to the patient at all hours of the day and night with a portable apparatus often arriving when the attack is over. However, I have done a great deal of this and I consider the time and the energy well spent, for the returns in diagnostic accuracy have been great. However, in order to facilitate the method I began some seventeen years ago to employ measures to precipitate attacks. The first of these was exercise. Of course exercise electrocardiograms are of no value unless the exercise is sufficiently vigorous. I have found 15 to 20 deep knee bends an excellent exercise for this purpose. It is to be remembered, however, that a normal exercise electrocardiogram alone does not rule out coronary disease. Later I learned that the characteristic electrocardiographic changes at times do not occur immediately after the exercise but may be delayed by some five to ten minutes or more, so that at the present time I make it a practice to repeat the after exercise electrocardiogram after an interval of five minutes and again at ten minutes in many cases.

Another method which I have used has consisted of reproducing, in the office whenever possible, the identical conditions under which the attacks occur spontaneously. For example, if the patient's attacks occur immediately after a meal, or upon walking after a meal, or upon lying down after a meal, I order lunch in the office for the patient and have him walk or lie down and electrocardiograms are made as soon as the discomfort begins and, or course, even if it fails to appear. In this manner I have been able to make electrocardiographic studies during attacks which otherwise could not have been made. At times even though the patient cannot be observed during an attack, or no sharp changes occur during an attack, serial tracings made over a period of a few days of recurring attacks will show progressive changes. This makes it unquestionable that the attacks are of coronary origin. An unchanging electrocardiogram even if abnormal does not warrant a conclusion.

In 1940, I added to these methods the

*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, Shreveport, April 30, 1952.

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anoxia test of Robert Levy but found it too time consuming and too cumbersome to justify the results obtained. I am now attempting to evaluate the ballistocardiogram as a method applicable to this problem.

At this point it is necessary to call attention to the fact that even over a period of several weeks' observation one may fail to have the opportunity of making electrocardiograms during an attack, one may fail to find electrocardiographic changes during an attack, one many fail to find electrocardiographic changes after exercise, or after reproducing the conditions under which the attacks occur, and one may find a normal ballistocardiogram, and yet the patient's attacks may still be proven to result from coronary insufficiency at subsequent studies.

Everyone will agree that it is desirable to avoid the delay of diagnosis as much as possible in these cases. The two, three, or four weeks of repeated study represents a most unsatisfactory period of anxious uncertainty for the patient and yet, for obvious reasons, one cannot make the diagnosis of coronary disease or rule it out lightly.

During the investigation of the many such cases which I have had the opportunity to study I have observed a few things which I feel are frequently of value in reaching a conclusion. These observations relate to the patient's description of his attacks.

First of all, it seems to me that the published descriptions of the attacks are woefully inadequate, in that they are much too limited in every way. Many cases are missed because coronary disease is not suspected in patients with pain in locations other than those which are commonly described.

SYMPTOMATOLOGY

I should like to record here the symptomatology of coronary insufficiency as I have seen it in patients with electrocardiographically proven coronary insufficiency.

The description of the discomfort is divided into four categories: (1) Quality. (2) Duration. (3) Location. (4) Circumstances under which it occurs.

The *quality* of the discomfort varies considerably. The following sensations are described in any of the locations discussed later: (1) pressure, (2) constriction, (3) dull ache, (4) burning, (5) numbness and tingling, (6) sharp pain. Any combination of these may occur. Common combinations are numbness and tingling accompanied shortly afterward by any of the other sensations. Anxiety, nausea, feeling like belching, sweating, and dizziness have accompanied any of the above sensations during an attack. Rarely is discomfort described as "sticking" in character, even in cases in which it is rather sharply localized.

LOCATION PATTERN OF PAIN

The *location* of the discomfort varies greatly from patient to patient. However, there is a marked tendency for the location of the discomfort to remain the same with every attack in the same patient, although it often *extends* to wider areas during prolonged attacks. The discomfort may be confined to the upper, mid, or lower sternal or epigastric regions. It may be confined to the little finger of either hand or the little finger of both hands and the same applies to the wrists, forearms, elbows, arms and shoulders. When in the shoulder the discomfort may be felt in the joint, or anterior to it or posterior to it. In the rest of the upper extremity it may be described as on the "inner" surface or deep, or the "outer" surface or all three. Frequently, discomfort in the extremities tends to spread proximally during the progress of an attack; e.g., pain in the wrist spreads to the forearms, then to the elbow, arm, shoulder, and finally, into the chest as the attack continues. In other cases the spread is in the reverse direction, pain beginning in the chest and spreading to the little finger. In its extension up or down the extremity or extremities the pain may skip over any of these locations; e.g., beginning in the chest it may then be felt next in the elbow, later in the forearm and wrist, skipping the shoulder and arm. Often when both extremities are involved, the manner of spread is symmetrical. However, one extremity, usually the left, often becomes

painful before the other and if the attack does not last the symmetry is not attained. The manner of spread of the location of the discomfort is usually characteristic for each patient.

The discomfort may be confined to the throat; to the lower jaw usually the left, or both lower jaws; to the ear, usually the left, or to both ears. Again, it may begin in the throat and spread up the neck to the jaw, or ear, or to the jaw and then to the ear. It may begin in the chest and spread to the neck and thence to the jaw and ear. In this spread it may skip regions as was described for the extremities. Also, as was described for the extremeities, it may begin in the more distal location (the ear) and reach the chest last.

The discomfort may be confined to the upper left pectoral area or to the lower left pectoral area. It may be confined to both upper or both lower pectoral areas. It may consist of a vicelike band of constriction about the whole chest at either the upper or lower levels. Less often it is confined to the right upper or lower pectoral area. As in other locations, it may begin on one side and extend to the opposite side, to the extremity areas, or to the head areas or to both, unilaterally or bilaterally.

The discomfort may be confined to the interscapular area, or beginning here may penetrate the chest to reach the sternal areas and thence elsewhere. Again, the reverse direction of spread is common. It may be confined to either or both scapular areas or suprascapular areas. Beginning here it spreads commonly to the extremity or extremities. Occasionally from the scapular area the pain spreads to the hypochondrium of the same side and to the shoulder area.

It is clear from the above that the pain of coronary insufficiency may occur in almost any part of the upper half of the body, either primarily or as immediate or later radiation. While this simple statement will hardly help a great deal in the recognition of this disorder a deeper realization of its truth may prevent us from making the errors involved in pronouncing too freely diagnoses such as globus hystericus, neuralgia, bursitis, cardiospasm, etc. Furthermore, in reviewing the great irregularity encountered in the quality and location of the symptoms of coronary insufficiency we encounter certain patterns which because they recur every now and again, deserve notice. I refer here to the recurrence in the above description of certain radiation patterns. In addition to the classic description of pain in the chest radiating to the left arm or both arms, there is the pain in the throat or neck with spread to the jaws, teeth and ears (in which any one or two of the named areas may be skipped). Seldom are paroxysmal attacks of pain following this pattern due to any disease but coronary insufficiency. Another phenomenon worth notice is the manner of progression of radiation of the pain during each attack which recurs with considerable regularity in the above discussion. Repeatedly, we encounter a regular distal or reverse progression of the "radiated" pain which takes place in stages (which may succeed one another with greater or lesser rapidity) during the progress of the attack and in a manner which tends to be characteristic for the individual. One is reminded of the spread of "radiated" pain from the lower quadrant to the renal, testicular, thigh, and knee areas which occurs as an attack of renal colic progresses. In the upper half of the body such stagelike progression of pain repeated regularly in paroxysmal attacks seldom occurs except in coronary insufficiency. And, although the locations may be bizarre, the stagelike progression during repeated paroxysms, particularly, if it is *regularly* from proximal areas to distal areas or in the reverse direction, is strongly suggestive of coronary insufficiency. I have not seen this phenomenon in diaphragmatic hernia, mediastinal disease, and spinal arthritis. Something close to it can occur as a hysterical conversion but here the progression of the discomfort is apt to be irregular instead of regular. Pain can occur and progress in a regular manner as a result of spastic colitis. The pain may begin in the right arm or shoulder, move to the mid-

sternum or manubrial area, then to the left shoulder and finally into the left arm. It is almost as if the radiation of the pain follows a perististalitic wave traversing the transverse colon from right to left. Most frequently in such attacks the pain in following this path moves as stated above; it does not spread to include more areas. However, occasionally it does spread and then we have a difficult distinction to make.

CIRCUMSTANCE PATTERN OF PAIN

The circumstances under which the discomfort of coronary insufficiency occurs also vary considerably, but here again there is a tendency for a particular pattern to be characteristic for the individual.

It is commonly stated that the discomfort frequently follows exertion and this is true. However, I should like to qualify this by reviewing certain observations which are interesting and which may be important diagnostically. Pain on walking one or two blocks is a fairly common complaint. If we crossexamine the patient we expect to find that similar or greater exertions other than walking also cause pain, but frequently we find an amazing incongruity. I have frequently seen persons who complain of pain on walking one half to one block and yet who have no difficulty in climbing stairs, and who say that they can do it "all day long". There is not time here for further illustrations but I have seen this disproportionate effect of various kinds of physical effort sufficiently often to be impressed by it and to avoid being misled by it. This is very easy to do if the other characteristics of the attacks are not "typical". Actually hysterical persons anxious about their hearts may develop pain following walking as well as any other kind of exertion; if they complain of pain on walking they will seldom fail to complain of pain on climbing a flight of stairs. Therefore, when a patient tells me he has pain on walking, but *not* on climbing stairs, I do not feel disposed to ascribe his complaints to anxiety.

It is important to note that persons who complain of pain on walking one block are frequently able to walk about the house or the office, or department store where they are employed without having attacks.

The attacks often occur following meals. This commonly occurs after a large meal. Some patients have attacks following meals, who do not have attacks on exertion and vice versa. The value of such histories in diagnosis is not so great. However, not too infrequently we find a patient who has no pain on eating, no pain on walking, but suffers pain if he walks after he eats. As a regularly recurring phenomenon I have not encountered this in any disease other than coronary insufficiency.

Another frequent circumstance under which the pain occurs is the assumption of the horizontal position. The patient complains that he gets an attack just as he lies down to go to sleep. Most frequently, the single attack occurs and once relieved the patient can lie down again and sleep all night. This is another phenomenon which does not occur regularly in conditions other than coronary insufficiency.

Again, occasionally a patient states that he does not have pain after he eats unless he lies down after eating. He has discovered that if he sits up for one-half to one hour after eating the attack can be avoided. The pain in such cases starts within a few seconds of assuming the horizontal position after eating a meal. Sitting up brings relief. This is another phenomenon which does not occur regularly in conditions other than coronary insufficiency, except possibly for occasional simulation by hiatus hernia. Lying down again brings the pain back. It is a reproducible phenomenon.

Emotional tension, best exemplified by sudden anger, is very commonly given by the patient as a circumstance under which attacks occur and this history is frequently furnished spontaneously. It can often be elicited that the attacks are more likely to occur with emotional upset following a meal than on an empty stomach. Continuing and unconscious tensions unquestionably increase the frequency of attacks under whatever circumstances they occur.

Occasionally pain occurs during sexual intercourse but this, in my experience, has

been an uncommon occurrence. I make it a practice to obtain information on this point in each case. Most who have pain on intercourse continue intercourse in spite of the pain, often after a brief pause. In my opinion the sexual act, though it may have fallen into disrepute in certain other respects, remains relatively innocent as regards the precipation of attacks of the pain of coronary insufficiency.

Attacks of pain occur commonly during sleep and usually awaken the patient. Actually sleep must be regarded as one of the commonest circumstances under which the pain occurs. The attacks during sleep are distinguished from the decubitus attacks by the fact that in those patients in whom they are frequent they occur even if the patient sleeps sitting up. In many such patients it is easy to prove that such pains occur at the climax of a particularly exciting dream. These attacks are, then, related to the emotional tension of the patient which, of course, is responsible for dreams of this kind.

The pattern may be only pain on walking or any one of the other circumstances above described. Commonly it consists of two or more, although one may be and often is quantitatively the more important. There is a tendency for the pattern of precipitating circumstances to remain characteristic for each case just as was found for the pattern of location.

DURATION OF PAIN

The duration of the discomfort in coronary insufficiency varies considerably. It may last, (without nitroglycerine) as long as several hours but generally when it occurs in repeated paroxysms it lasts a much shorter period of time, seldom longer than thirty to forty minutes. If pain characteristically occurs during a certain activity, such as walking, its duration may be determined by the patient's decision as regards the continuation of that activity. Generally, when the activity is stopped, discomfort begins to subside and may be gone within a minute. Occasionally, it lasts several minutes. If the activity is resumed the discomfort may return in the same period of time that it required for its development before. At times, it requires progressively less time, and occasionally, paradoxically, it does not return even with a prolonged period of the same activity. At times, the discomfort disappears even if the activity which precipitated it is not discontinued at all (even without the use of nitroglycerine).

Although in most cases the attacks last from three to twenty minutes, it is not rare for them to last only one to two minutes or less. I have seen one patient whose attacks lasted eight to ten seconds.

There is a tendency for the duration of the attacks to remain the same for each patient.

Then, we find in studying the circumstances under which the symptoms occur and their duration a wide variability, embracing even certain phenomena which appear incongruous and paradoxical. Our recognition of this variability and of the incongruous and paradoxical behavior may prevent us from falling into the error which is made through the expectation of a more classical and more understandable response to exercise, rest and other circumstances. Furthermore here again, we find amidst a wide variability certain recurring patterns and characteristics other than the classical which deserve notice. The patterns deserving notice have been pointed out already, but I should like to call attention to the observation that there is a tendency for the various patterns to remain constant, the location pattern, the circumstance pattern and the duration all tend to remain constant in each case. It is these phenomena which in many cases lead to the recognition of coronary insufficiency. A person may have pains in the chest; he may have pains in the chest after various exercises; and he may have pains in the chest after exercise which lasts anywhere from five to twenty minutes. Such a patient may or may not suffer with coronary insufficiency. But if a patient has pains in the chest which are always in or begin in the *same area*, even if it is the suprascapular region, and if it usually comes on after the same exercise,

as when walking to the carline, and they always last four or five minutes, then he almost certainly has coronary insufficiency. If in addition, there is a spread to other areas in the same manner in each attack the evidence is even stronger. In other words, the tendency toward constancy of the location and of the circumstances, and to some extent, of the duration of the pain should help considerably in the recognition of the disease regardless of how bizarre or atypical the location, the circumstances, and the duration may be. One patient had pain only in the throat, only on cleaning the table after mid-day meal, and it usually lasted ten minutes. These phenomena occur in no other disease with which I am familiar. Of course, it is important to remember that many cases do not exhibit this regularly. Its absence does not rule out coronary insufficiency.

SUMMARY AND CONCLUSIONS

1. The symptomatology of coronary insufficiency has been described as it has been encountered by the author.

2. Paroxysmal attacks of pain occuring in the upper half of the body, which in every attack begin in the same area, *and* which in every attack progress in stages to other areas which are also always the same and are in every attack reached in the same order, are almost certainly due to coronary insufficiency, regardless of how "atypical" in location these areas may be. If in addition, the attacks are relieved within 3 to 4 minutes by nitroglycerine the diagnosis is unquestionable. Gall bladder colic is the only exception to this rule and it does not respond dramatically to nitroglycerine.

3. Pain in the chest or epigastrium which does not occur simply on walking or after a meal, but which does occur on walking after a meal is presumably due to coronary insufficiency. Response to nitroglycerine makes the diagnosis certain.

4. Pain in the chest, mid epigastrium, both arms, or left arm on lying down after a meal which does not occur if patient sits up after a meal is almost certainly due to coronary insufficiency. If the patient does not have a diaphragmatic hernia the diagnosis is unquestionable.

5. Pain occuring regularly when patient first (within a few seconds to three minutes) lies down in bed at night is presumptive evidence of coronary insufficiency. Response to nitroglycerine or avoidance by taking nitroglycerine just before going to bed makes the diagnosis unquestionable.

I should like to apologize for having the temerity in this day and age to present a paper which pretends that history taking may at times yield more information than a gadget or a laboratory test.

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POSTCORONARY SYNDROMES*

BERNARD M. KALSTONE, M. D.
SHREVEPORT

This paper will not be an attempt to discuss every complication or aftermath of an attack of coronary disease but will be confined to two syndromes. One is well known and is labeled the "shoulder-hand syndrome". The second condition, although I am certain that all of you are aware of it, has rarely been discussed in detail in the literature. Yet it is a most practical one as it deals with the patient's ability to earn a livelihood following a heart attack. It is chiefly a symptom complex which Flaxman[1] termed the "postcoronary syndrome."

SHOULDER-HAND SYNDROME

The shoulder-hand syndrome is a complication that usually develops three to four weeks following an acute coronary attack. In practically all cases this comes after a myocardial infarction rather than an uncomplicated coronary insufficiency. This is not an absolute prerequisite, however, as it has been reported following severe coronary disease without clinical or laboratory manifestations of an infarct.[2] These individuals complain of pain usually in the shoulder with radiation to the hand or to a concomitant pain in the hand. Many patients when the persistent pain develops in the shoulder, first interpret the symptom as

*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, Shreveport, April 30, 1952.

having resulted from some further injury to the heart.[3] The shoulder disability most often precedes that of the hand. The left shoulder is the one generally involved. In some cases the pain alternates from one shoulder to the other.[4] The pain has been described variously as burning or aching or wrenching. Frequently, there is a trigger point or zone on the shoulder or over the scapula. Pressure applied to this area will reproduce the symptoms complained of by the patient. The pain is usually continuous often with exacerbations and attenuations. Whenever the hand is affected, in addition to the presence of pain, the hand is generally swollen and frequently discolored. The average case develops no further than this, and then gradually, from a few weeks to several years, makes a complete recovery. Usually the shoulder pain leaves first with slower improvement of the hand.[5]

There are some unfortunates that go on to a more advanced stage characterized by limited and painful movements of the fingers and a contracted shoulder. The muscles become atrophic and the bones of the hand or of the shoulder-end of the humerus may exhibit trophic changes on x-ray. Some individuals in this stage may yet have a partially reversible pathology.[2] Most of those, however, who are hapless enough to reach this stage have a permanent disability with crippling of the shoulder and a contracted, clawlike hand. A Dupuytren-like contracture may result.[6] Although remarkable trophic changes are apparent, the pulsations of the arteries of the affected extremities are normal. Trophic ulcers of the fingers[7] and a "sclerodactylic" hand[8] have been reported.

If the pain of the myocardial infarct does radiate to a particular shoulder or arm, it does not necessarily predispose that same side if the shoulder-hand syndrome later develops. The occurrence of this syndrome bears no relationship to the anatomic location of the myocardial infarct. Hilker[2] feels that there is a direct ratio between the severity of the infarction and the development of this reflex-dystrophy and further, that it develops only in those with left heart failure and prolonged hypotension. He also states that if the myocardium heals, the syndrome is interrupted, but if the myocardium remains incompetent, the shoulder-hand effects will continue.

The etiology of the shoulder-hand syndrome is not completely understood. The most favored theory is concerned with a disturbance of the so-called internuncial pool. This pool as described by Steinbrocker *et al.*[9] consists of an extensive network of interconnecting neurones in the central gray matter extending over many segments of the spinal cord. At these levels potential connecting pathways are formed between incoming impulses and motor neurones of either the sympathetic (posterolateral) or the anterior horn cells. The afferent stimuli arise from an area of injury, either trauma or a physiologic irritation. In the case of a myocardial infarction these stimuli are carried by the cardiac nerves and enter the spinal cord at the level of T_1 to T_4 (Figure 1). Here they stimulate the internuncial pool of those segments. The anterior horn cells are affected and a disability of the shoulder muscles ensues. The lateral horn cells are also irritated with resultant changes in the fingers and

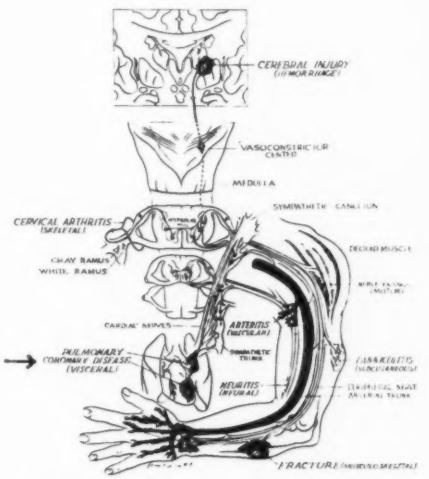

FIG. 1. A diagrammatic representation of current neurophysiologic concepts applied to the shoulder-hand syndrome and some of its causes.

(*After Steinbrocker et al*[9]).

hand. As has been pointed out[9] there must be continuous activity of the internuncial pool with a self-exciting, self-perpetuating mechanism. Anoxia with defective local tissue nutrition might be the stimulus that prolongs the condition and its subsequent correction would then be responsible for interrupting this cycle and reversing the process.[2] The osteoporosis that develops is that type of bone atrophy that follows a prolonged, deep hyperemia. Those interested in the etiology of this syndrome admit that the aforementioned theory does not answer all questions but that it appears to best fit all the facets of the shoulder-hand syndrome.

The treatment of the shoulder-hand syndrome in brief is: (1) Physiotherapy—this is strictly palliative; (2) Radiation; this is usually ineffective. (3) Block therapy. Paravertebral and brachial plexus block have been beneficial in some cases. The shoulder disability is more apt to respond than is that of the affection of the hands. Sympathetic block in the presence of a myocardial infarction, however, is not without danger. (4) Surgery has been used with moderate success, the procedures varying from periarterial sympathectomy to chordotomy.

Before concluding the discussion of this syndrome, it should be mentioned that it appears unlikely that the prolonged period of absolute bed rest enforced by many physicians in the treatment of coronary disease has any bearing upon the subsequent development of the shoulder-hand syndrome. This syndrome is not one of the evils of strict bed rest which were so well presented by Dr. Samuel Levine.[10]

SECOND SYNDROME

The second syndrome to be discussed occurs in the so-called recovery stage following coronary disease. This is the period when the patient begins to be more active physically or perhaps when he first returns to work. This syndrome is not as generally recognized as is the shoulder-hand syndrome. It is chiefly concerned with a series of peculiar chest sensations. The most common of these, in my experience, is a feeling of the patient whereby he claims that he cannot get his breath below a certain level of his chest, usually indicating the third interspace of the anterior chest as the lowest point that air will reach. This is often associated with a "tight feeling" of the thorax and oftentimes the patient declares that the sensation resembles that of a band drawn taut around the chest. The soreness or tightness of the chest is not relieved by nitroglycerin. The aforementioned symptoms are not unlike those complained of by anxious individuals who are unknowingly hyperventilating. Fatigue and weakness are prominent complaints. These symptoms are representative of a critical stage in the recovery of the patient as he begins to fear that he will never again be able to do a day's work. He also fears that his discomfort is a permanent one. Consequently, it is important for the physician to recognize his plight and to offer the patient the firm reassurance that it is only a temporary phase in his postcoronary course. Time and continued activity will see the end of this peculiar feeling in his chest. Do not put the patient back to bed or markedly restrict his activity at this point. Give him repeated reassurance. You must point out to the individual that he is not experiencing true angina nor is this a warning of impending coronary occlusion. It is simply a common postcoronary occurrence that will disappear.

Failure to appreciate the significance of this syndrome and, more especially, failure to reassure the patient and to adequately explain his symptoms may inadvertently lay the ground work for a pronounced cardiac neurosis. Advise the patient to continue his activities and remove the constant dread that his symptoms may be due to a permanently damaged heart.

In a study made by us[11] of 120 cardiacs who survived an acute attack of coronary disease we found that after a reasonable convalescence, severe restriction of physical activity beyond the requirement imposed by limited cardiac cardiac reserve affords little or no protection to the patient

who has survived the acute attack. (Figure 2).

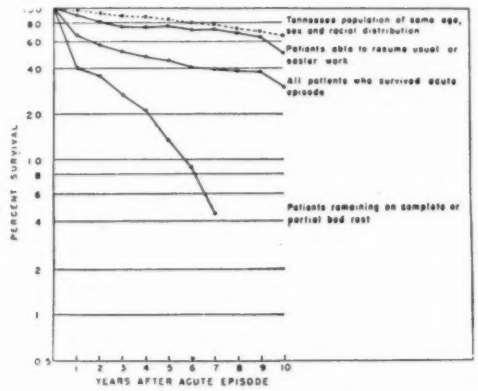
Tennessee population of same age, sex and racial distribution
Patients able to resume usual or easier work
All patients who survived acute episode
Patients remaining on complete or partial bed rest
PERCENT SURVIVAL
YEARS AFTER ACUTE EPISODE

Figure 2.—Myocardial infarction — Survival rates; survival curves plotted on logarithmic scale. The slope of a line segment is a measure of the mortality rate during the period in question. Parallel slopes indicate similar mortality rates. Computed from mortality rates published by U. S. Bureau of the Census. (*After Billings et al*[11]).

It is advisable for the physician not to be too free in the recommendation to the patient that he quit work permanently or for a prolonged period subsequent to a coronary attack. Unless that person has a very poor cardiac reserve or severe angina he will likely be able to return to his former activity. Be optimistic to the patient and give him encouragement in the face of this postcoronary syndrome. Reassurance at this stage in the patient's recovery can assist him in making a satisfactory adjustment to his situation. In a paper several years ago, Auerback and Gliebe[12] discussed iatrogenic heart disease, that is, heart disease produced by doctors. A patient exhibiting anxiety symptoms can very easily become a cardiac neurotic because of a careless phrase or a mild warning of his physician. It is the same with these "postcoronary" patients. They can be transformed into lifelong invalids if at this stage in their postcoronary recovery thay are not encouraged concerning the temporary and relatively mild nature of their complaints. It must be mentioned that definite cardiac failure can occur and the recognition of the postcoronary syndrome must not blind one to the possibility of true cardiac failure or of poor cardiac reserve.

SUMMARY

The shoulder-hand syndrome frequently follows a myocardial infarction. Most cases eventually recover but a few go on to permanent atrophy of the hand or shoulder. This syndrome bears no relationship to the radiation of the pain or to the anatomic site of the causal myocardial infarction. The mechanism of the shoulder-hand syndrome is best explained on the theory of traumatic cardiac stimulation to the spinal cord through an internuncial pool and via the cardiac nerves. This initiates a vicious circle resulting in a reflex-dystrophy that is probably maintained by the anoxia and the defective local nutritional status of the heart muscle. The syndrome apparently is not caused by the prolonged bed rest with limitation of motion of the extremities in the treatment of the coronary attack.

A second postcoronary syndrome has been described. This is characterized by tightness of the chest, difficulty in breathing and weakness. The syndrome occurs when the patient attempts to resume his usual activities. The symptoms tend to disappear as the individual continues his work. Wholehearted reassurance to the patient is most necessary at this stage of his postcoronary recovery and continued activity should be encouraged and not discouraged. The outlook for the patient with this postcoronary syndrome is good.

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DIAPHRAGMATIC HERNIA SIMULATING THE PAIN OF HEART DISEASE*

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SHREVEPORT

There have been several articles[1-5] in recent medical literature discussing the importance of diaphragmatic hernia in the differential diagnosis of coronary artery pain. We wish to report a few of the patients seen in our office over the period of the last two years which emphasized this diagnostic point.

All of these patients were either referred to us, or came to us, with a diagnosis of coronary artery disease. We were called out to see several during an acute attack and hospitalized them for observation for coronary occlusion. All of the patients had complete histories, physical examination, and laboratory studies, including hemogram, urinalysis, and serology. Electrocardiograms were taken repeatedly on all patients and usually the exercise test was done with tracings taken before and after the test. Gastrointestinal roentgenograms were done by two of us (G. M. R. and W. H. C.). In every case, during fluoroscopy, the patient was placed in the Trendelenburg position and the Valsalva maneuver was performed immediately after swallowing barium.

*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, Shreveport, April 29, 1952.

CASE REPORTS

Case No. 1: White female, age 65. This patient suddenly developed severe pain in the substernal region which radiated to her left shoulder. There was no dyspnea or shock. The blood pressure remained at 160/90. An opiate was given. She was hospitalized and repeated electrocardiograms were negative. The history revealed that she had had recurrent similar attacks of substernal pain radiating down the left arm occurring usually after exertion, but frequently at night. She had been told she had heart disease by a physician, but there were never any electrocardiographic findings. She learned by experience that if she ate very lightly for her evening meal she would not have an attack at night. She was discharged. Following

Figure 1—Case No. 1

this, she had many similar attacks that usually required morphine, demerol, or pantapon for relief. There was never any shock or change in blood pressure. She had had two roentgenological studies of the gastrointestinal tract and these were reported completely negative. She finally complained of difficulty in swallowing. She stated that there was a lump present in her substernal region every time she would swallow. On August 12, 1949, a recheck gastrointestinal series was done, and a hiatus hernia of moderate size was found. Since that time she has been on antispasmodics, sleeping in a semireclining position, and has been advised not to eat for several hours before retiring for the night. Repeated electrocardiograms have still been normal. She has had occasional pain, but now she does not become alarmed about heart disease whenever she has it. Occasionally, when she is lying down flat, she has substernal pain.

Case No. 2: White male, age 28, physician. This patient gave a history of having had an appendectomy in December, 1943, while he was in the Navy. This was followed by a pulmonary embolus. A short while later he developed pain starting in the substernal region, radiating up the left shoulder after exertion. This was considered secondary to precordial adhesions. Repeated electrocardiograms and x-rays of the chest were completely normal. He continued to have pain on exertion; or mild activity, particularly after a full meal, would bring on the pain. He developed hepatitis in May, 1949, and recovered. He had a recurrence of his hepatitis in March, 1950. Icterus index was 11.6.

Figure 2—Case No. 2

There was slight tenderness in the epigastrium. Hemogram and urinalysis were normal. A cholecystogram was done later, which was normal. A gastrointestinal series was done and a large diaphragmatic hernia was found. After adequate rest he recovered from his hepatitis. It was felt that his substernal pain was secondary to his diaphragmatic hernia. He understood the situation and stated that he did not wish to have recurrent severe pain in the heart region, and, therefore, requested repair. A transthoracic surgical repair was done. He had a very smooth postoperative course. There have been no further symptoms of substernal or cardiac pain.

Case No. 3: While male, age 66, priest. This patient gave a history of having had recurrent substernal pain, radiating up the left shoulder whenever he would walk up a hill after a full meal. This had been coming intermittently for the past few months. History revealed that he was able to rake his yard and work without any pain, but as soon as he would walk after a full meal he would develop the attack. Nitroglycerine did not seem to relieve it. He was obese. He had no other digestive symptoms, shortness of breath or any cardiovascular symptoms. Physical examination revealed a well developed, obese, short sthenic individual. Examination of eyes, ears, nose and throat was essentially negative. Blood pressure was 120/80. Examination of the heart and lungs was normal. Repeated electrocardiograms were taken and revealed no abnormalities whatsoever. Gastrointestinal series revealed a fair sized diaphragmatic hernia. He was placed on a bland diet with antispasmodics and told to sleep in a semi-reclining position. Since that time he has had no pain whatsoever.

Case No. 4: A white male, age 39, oil field worker, was seen in November, 1950, complaining of pain around the heart. This was characterized as intermittent in character, located in the substernal region and radiating through to the back. There was no shock associated with it. The pain would last a few minutes and then disappear. Activity would not usually bring it on. The patient had had morning vomiting for ten years. Physical examination was negative. Hemogram and urinalysis were negative. An electrocardiogram was completely normal. Roentgenographic views of the chest revealed peribronchial thickening in both lung fields. Studies of the gastrointestinal tract revealed a hiatus hernia with an ulcer. He was placed on supportive medication of antispasmodic therapy and diet and the symptoms cleared.

Case No. 5: A white female, age 42, housewife. The patient had had frequent attacks of substernal pain, radiating up behind the sternum. It came on at various intervals, with or without exercise, and not particularly associated with eating. She felt she had heart disease. She had had repeated electrocardiograms taken and they were always negative. In September 1951, she suddenly developed a severe gastrointestinal hemorrhage, requiring transfusions. The substernal pain was present at this time. She was hospitalized and an electrocardiogram was negative. After one week, a gastrointestinal series revealed a diaphragmatic hernia. After being placed on symptomatic treatment and a bland diet, she has had no further so-called "heart pains."

Case No. 6: A 48 year old white male, a grocer, gave a history of having had intermittent pain in the left upper quadrant, radiating up the left shoulder and down the left arm of several weeks' duration. He stated it felt like gas and came on after he ate. Sweet milk and belching seemed to relieve it. Occasionally he noticed it when he would lie down at night. Often the pain would

start in the left costal margin and spread up the left shoulder and down the medial aspect and the ulnar region of the left arm. There was no shock connected with the pain. Physical examination revealed a well developed, well nourished individual, with no indigestion. Blood pressure was 130/80. Heart and lungs were negative. There was slight tenderness in the left costal margin. The liver was just palpable. Repeated electrocardiograms were completely negative. A gastrointestinal series revealed a small hiatus hernia. After giving him antispasmodics and a bland diet and advising him not to eat at night, his pain subsided. He has had no further pain.

Case No. 7: A white male, age 65, businessman, gave a history of recurrent attacks of rather severe pain in the left upper quadrant and the left lower chest. This was severe and constant and became localized at the apex of the heart. This usually occurred at night. There seemed to be no particular relation to exercise or to eating. There was no shock. Physical examination revealed a slender, asthenic individual. The heart and lungs were essentially negative. The blood pressure was 130/80. The liver was palpable but not tender. Roentgenograms of the chest revealed a thickened pleura at both bases with bronchiectasis at the right base. There was a large esophageal hiatus hernia. The stomach and the remainder of the gastrointestinal series and barium enema were negative. He was advised to rest in a semisitting position, given antispasmodics, placed on a bland diet and he had no further symptoms.

Case No. 8: A 65 year old white male, executive, was seen on November 5, 1951, with severe pain over the xiphoid process. He had been out at his camp on a Sunday and after dinner developed a severe pain below the xiphoid process. It was acutely severe, lasted about an hour, after that it cleared. Following this it again recurred and he appeared in a shocklike state. He came to the hospital. No particular abnormalities were found on examination except for pain in the lower substernal region. Electrocardiogram was taken, which was normal. There was no temperature elevation or shocklike state. He was rechecked the following day. At this time the abdomen was soft and there was no evidence of any blood pressure changes. A gastrointestinal series revealed an enormous hernia of the esophogeal hiatus, slight deformity of the duodenal cap with no evidence of an ulcer. There was some spasm throughout the colon with irregularity of the sigmoid colon with small diverticula present. He was put on antispasmotics and a bland diet, and told to sleep in a semisitting position. He has been essentially symptom free since that time.

Case No. 9: A white male, salesman, age 34, was seen on August 25, 1951. He gave a history of having epigastric fullness for several weeks prior to this, with pain in the left lower chest, usually after driving a long period of time and after excessive fatigue. This would produce a tachycardia and aggravate his nervousness. He also developed tightness in the neck with a pulling pain in the left shoulder. Further history revealed that he had bloating and pain in the left pectoral region after eating. Physical examination revealed a well developed, slightly obese individual with a blood pressure of 120/80. Electrocardiogram was repeatedly negative. A gastrointestinal series revealed a small esophageal hiatus hernia, no other significant abnormalities. There were diverticula in the colon. He is essentially symptom free on supportive treatment.

Case No. 10: A 35 year old white female nurse gave a history of having recurrent attacks of substernal pain, well localized, occurring after eating. It would last a few minutes and then disappear. There were no other cardiovascular symptoms. Examination revealed a slender, asthenic individual with no other particular abnormalities. An electrocardiogram was negative. A gastrointestinal series revealed a small hiatus hernia. The patient was placed on antispasmodics and small feeding and the symptoms cleared.

Case No. 11: A white male dentist, age 55, gave a history of having had recurrent substernal pain occurring after he would walk to his quarters after a full meal. This became progressively worse and he felt he had heart disease. Electrocardiograms were taken and were completely normal. A diaphragmatic hernia was found on x-ray. He was seen three years later and had no further symptoms except when he would overload his stomach.

Case No. 12: A white male farmer, 55 years old, complained of severe recurring attacks of pain to the right of the sternum of one week's duration. He had been a hypertensive for years and thought that this was his heart. There was no aggravation with exercise. He had had indigestion for the past few years. Physical examination revealed a well developed, obese, hypersthenic individual with a blood pressure of 180/90. Repeated electrocardiograms were normal. Roentgenogram of the chest revealed a transverse heart with no definite enlargement. A gastrointestinal series revealed a small diaphragmatic hiatus hernia. Gall bladder series was negative. He has been asymptomatic since then on supportive therapy.

Case No. 13: A white male, age 40, refinery worker, was seen on May 4, 1950. He gave a history of having had pain in the epigastrium, intermittently since 1936, relieved by milk. Physical examination revealed a tall, asthenic individual with tenderness in the epigastrium. The remainder of the examination was negative. A gastrointestinal series revealed definite deformity in the region of the bulb, which was believed to be residual scarring due to an ulcer. His symptoms

improved on banthine and he was symptom free for several months. He was again seen eighteen months later with similar pain. At this time he also complained of some pain in the chest and substernal region. He was worried about his heart at that time. Electrocardiogram had been completely normal. A gastrointestinal series revealed a moderate sized hiatus hernia, slight deformity of the duodenal cap. X-ray of the chest was essentially negative. There was thickened pleura at the right base. He is again asymptomatic on banthine, sedatives, and a bland diet.

Case No. 14: A white male refinery worker, age 45, complained of precordial fullness and pain for the past five years which came on suddenly, associated with the feeling that he could not catch his breath. This would last one-half to one and a half hours and occurred two to three times a week. Most of the symptoms occurred at night. An electrocardiogram was taken and revealed a midline heart, no evidence of ischemia or any other abnormalities. A gastrointestinal series revealed a small hiatus hernia. He was placed on sedatives and antispasmodics and reassured that he did not have heart trouble.

Fourteen patients have been reported in detail, and 13 others have been found which have been diagnosed as coronary artery disease but the pain was actually due to diaphragmatic hernia. Careful history will usually reveal that the pain usually occurs at rest and after a full meal. Sitting in a cramped position, such as in driving a car, or wearing a tight abdominal binder, usually precipitates the pain. The majority of patients complained of pain occurring whenever they would lie down and turn on the left side. Exercise does not usually bring on the pain, but it may aggravate it after a heavy meal.

The majority of the patients are males, hypersthenic and obese and between 23 and 73 years of age. Electrocardiographic changes attributed to the hiatus hernia consisted of transient low T-waves in lead 1. These were interpreted as due to temporary local ischemia due to vasospasm due to pressure. No electrocardiographic changes were ever observed after exercise tests. Associated symptoms of indigestion were present in many patients. Three patients were subjected to surgical correction of the defect. The symptoms have not recurred. The majority of patients can be treated medically by weight loss, antispasmodics, small meals, and lying in a semisitting position.

CONCLUSIONS

Esophageal hiatal hernia can produce symptoms similar to those due to coronary disease. Fluoroscopic study for this defect should be an integral part of a diagnostic study for any type of substernal pain. The presence of a hiatal hernia does not in itself signify that it does cause the substernal pain. Careful history and laboratory study must be done along with clinical observation in order to arrive at the correct etiological factor.

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THE CARDIAC PATIENT AS A SURGICAL RISK*

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NEW ORLEANS

The surgical risk in cardiac patients is probably as old a problem as surgery itself. So much so, in fact, that fear of the heart is still often based more on tradition than on established scientific fact. There still exists in some quarters an unwarranted fear of performing any kind of surgery on people with heart disease. Although there are relatively few comprehensive statistical studies on the subject, practically all investigators have reached optimistic conclusions

*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, Shreveport, April 29, 1952.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the results of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

regarding the ability of the heart to withstand anesthesia and surgery. One of the earliest surveys of this sort was done by Butler, Feeney, and Levine, in 1930, who reviewed 414 cases subjected to 494 operations. Their mortality at that time was 6.3 per cent, which led to the conclusion that, as a group, patients with heart disease undergo surgery fairly well. Since that time other authors have repeatedly shown that an anesthetic or surgical procedure imposes little or no added burden on the heart; and if patients with heart disease are properly evaluated and managed, they can undergo major surgical procedures with little or no added risk. As a matter of fact, known cardiac patients often receive more attention than usual and may even be "especially safe because they are considered especially unsafe."

In recent years the mortality and morbidity rate of patients with heart disease who undergo surgery has been on the decline. This is due in part to the closer collaboration among internist, anesthetist, and surgeon. As evidence of this fact, 80 per cent of all patients in this study were seen in consultation or were studied by the medical service in preparation for surgery. It has been stated that the choice of anesthetist is of greater importance than the anesthetic agent—a truism which can hardly be denied. With improvement in technique of administration of anesthetic agents, the skill of the anesthetist is added to the calculated appraisal and preoperative preparation of the cardiac patient by the internist. The triad is completed by the surgeon who performs his task with a minimum of delay and who exercises more than usual precautions in avoiding shock and anoxemia.

The internist is often placed in the position of rendering an opinion as to whether or not a given patient is able to survive an operation. He should be able to give an intelligent answer to such a request—neither denying surgery to all patients with heart disease nor permitting surgery on everyone without consideration of the many factors that may alter the outcome. We have adopted a liberal policy based on earlier experience and on observation at the Veterans Administration Hospital since 1946. Very few patients who require surgery are refused, although they may have what some authors consider to be absolute contraindications. Actually, there are very few surgical conditions that cannot wait a few hours during which time the cardiac status can be evaluated and properly managed. With the advent of rapidly acting digitalis or strophanthin preparations and the availability of intravenous or intramuscular quinidine or pronestyl, congestive failure and cardiac arrhythmias can usually be controlled in a very short time. There are, as far as we are concerned, no absolute contraindications to emergency surgery and few, if any, contraindications to elective surgery. It must be remembered that every operation carries with it a certain element of danger and that not every patient who dies on the operating table or shortly thereafter does so because of heart disease.

It is true that the heart is involved in every death—the cessation of the heart beat is the ultimate criterion of death—but the heart need not be implicated in every postoperative fatality. It is with this thought in mind that we emphasize that these statistics are not inflexible and are therefore subject to appraisal on an approximate basis. No statistics can include such intangibles as the will to live or die, or measure the human frailties that may mean the difference between life and death. We offer these figures as a fact accomplished for whatever they are worth.

This particular study was undertaken primarily to determine the mortality rate of cardiac patients operated upon at the Veterans Administration Hospital at New Orleans during the past six years and to determine what factors, if any, influenced significantly the mortality and morbidity rate.

There were 500 operations performed on 334 patients. Twenty-three of these patients died, giving a total mortality of 4.6 per cent. Nine of these died an "inevitable" death, that is, patients who would have died in a reasonably short time if surgery had

not been performed and in whose death the cardiac factor played a secondary role. For example, a palliative colostomy or jejunostomy in a patient with inoperable carcinoma who died two to three weeks later is considered an inevitable death. Fourteen of the patients died an "unexpected" death, that is, patients who died or whose lives were shortened as a result of the operation. The unexpected mortality rate was a low 2.8 per cent. All future references in this paper apply to unexpected deaths unless otherwise specified. A further analysis of the mortality rate for the different types of heart disease may be found in Figure 1.

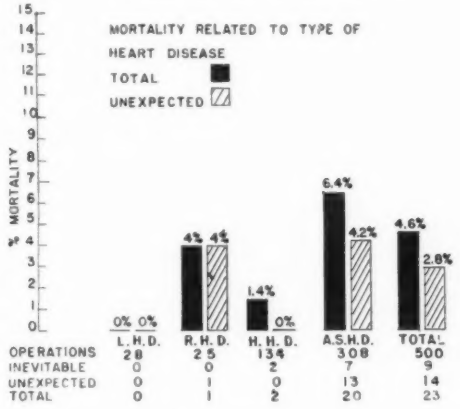

Figure 1

MORTALITY RELATED TO TYPE OF HEART DISEASE

There were 28 operations on patients with syphilitic heart disease and no deaths —a mortality rate of zero. There were no complications in this group. Syphilitic aortitis, aortic regurgitation, and aortitis with angina have been said to increase the risk considerably. All of these patients had aortitis with aortic regurgitation, and 5 had angina. This is admittedly a small number, but nevertheless, none died.

Twenty-five operations were performed on patients with rheumatic heart disease with 1 unexpected death, a mortality rate of 4 per cent. This patient expired on the second postoperative day of atelectasis, pneumonia, and bilateral hydrothorax following a thoracotomy for bronchogenic carcinoma. The procedure lasted over two hours and was performed with cyclopropane anesthesia.

In the hypertensive group, 139 operations resulted in 2 inevitable deaths with a mortality rate of 1.4 per cent and no unexpected deaths—a mortality rate naturally of zero. This supports the almost unanimous opinion that hypertension per se contributes nothing to the element of risk.

The arteriosclerotic group comprised the largest number of operations and resulted in the greatest number of deaths. Arteriosclerotic heart disease understandably presents the gravest risk of all forms of heart disease. It occurs in the older age group, arrhythmias are often irreversible, congestive heart failure is present in large number, and the surgical condition is often of a malignant nature. Furthermore, it is difficult, if not sometimes impossible, to make an accurate diagnosis of coronary arteriosclerosis. In spite of statements to the contrary, not many patients die of a coronary occlusion following operation. Only 1 of the 308 patients with arteriosclerotic heart disease out of the entire 500 operations in this study resulted in death due to coronary occlusion. In 308 operations there were 20 deaths with a total mortality of 6.4 per cent. Thirteen of these deaths were unexpected, giving a mortality of 4.2 per cent. Three were due to congestive heart failure, 2 were due to shock and pneumonia, 1 each was due to cerebrovascular accident, pulmonary embolism, cardiac arrest, and coronary occlusion. One death was unexplained.

The total mortality in 500 operations on patients with all types of heart disease was 4.6 per cent. The unexpected mortality was 2.8 per cent.

The presence of hypertension in patients with arteriosclerotic heart disease increased the mortality rate slightly but not significantly (Figure 2). There were 197 operations on patients with arteriosclerotic heart disease without associated hypertension resulting in 7 deaths, a mortality of 3.6 per cent. There were 6 deaths in 111 operations in patients with arteriosclerotic heart disease with associated hypertension, resulting in a mortality of 5.4 per cent.

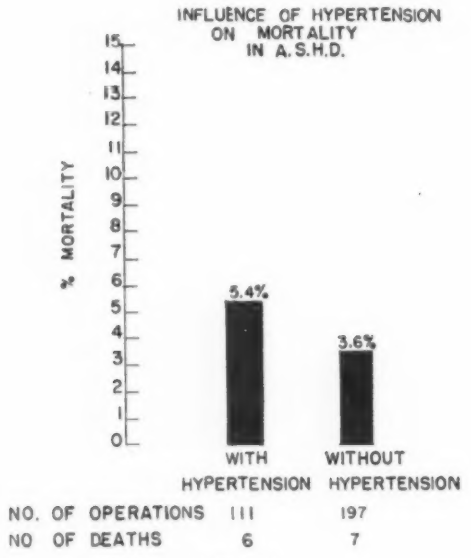

Figure 2

MORTALITY RELATED TO AGE AND RACE

All of these patients were males over 20 years of age, the majority being between 50 and 70, although 60 patients were over 70 years of age (Figure 3). There were 2

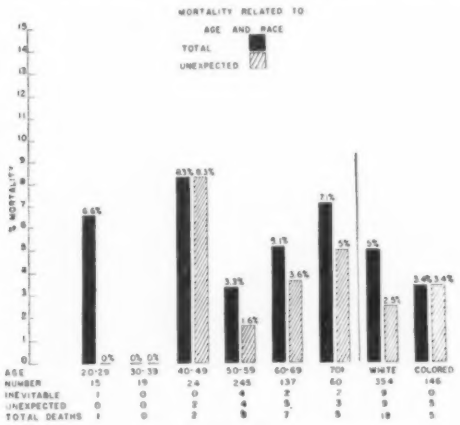
MORTALITY RELATED TO AGE AND RACE
TOTAL
UNEXPECTED
% MORTALITY
AGE 20-29 30-39 40-49 50-59 60-69 70+ WHITE COLORED
NUMBER 15 19 24 245 137 60 354 146
INEVITABLE 1 0 0 4 7 7 9 0
UNEXPECTED 0 0 2 4 5 3 9 5
TOTAL DEATHS 1 0 2 8 7 5 18 5

Figure 3

deaths in the 40 to 49 group with a mortality of 8.3 per cent; except for this, there was a progressive increase in mortality with age. There was less than 1 per cent difference in mortality between white and colored patients . There was no opportunity to study the difference in mortality according to sex; but in a previous unpublished study by one of us (J.E.G.), it was shown that colored females withstand surgery best, followed by white females, with white and colored males being about equal.

MORTALITY RELATED TO ORGANIC CARDIAC DEFECTS

In an effort to determine the effect of various organic cardiac abnormalities on the mortality rate, the influence of congestive heart failure, murmurs, cardiac enlargement, conduction defects, the presence of angina or previous coronary thrombosis, and the functional classification were all separately evaluated (Figure 4).

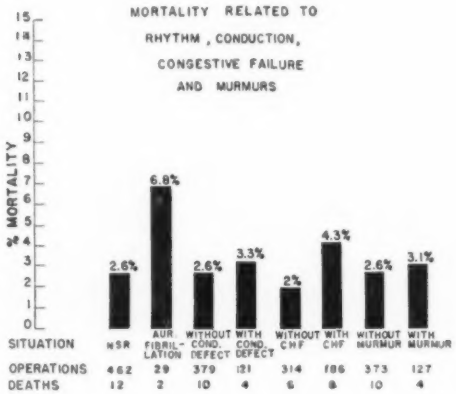
MORTALITY RELATED TO RHYTHM, CONDUCTION, CONGESTIVE FAILURE AND MURMURS
% MORTALITY
2.6%
6.8%
2.6%
3.3%
2%
4.3%
2.6%
3.1%
SITUATION NSR AUR. FIBRILLATION WITHOUT COND. DEFECT WITH COND. DEFECT WITHOUT CHF WITH CHF WITHOUT MURMUR WITH MURMUR
OPERATIONS 462 29 379 121 314 186 373 127
DEATHS 12 2 10 4 6 8 10 4

Figure 4

AURICULAR FIBRILLATION

In 29 patients operated upon with auricular fibrillation there were 2 deaths, a mortality of 6.8 per cent compared to 2.6 per cent of 462 cases with normal sinus rhythm. Auricular fibrillation thus can be seen to alter significantly the mortality rate. Even though this total number was rather small, other investigators are also cautious in regard to fibrillation and would prefer reversion to sinus rhythm before surgery if possible or reduction of the ventricular rate to 90 or less. Auricular fibrillation should not per se contraindicate surgery.

CONDUCTION DEFECTS

Of 121 patients with conduction defects (bundle branch block, delayed intraventricular conduction, and auriculoventricular block), only 4 died—a mortality of 3.3 per cent compared to 2.6 per cent mortality in 379 cases with normal conduction. It thus can be said that in this series conduction de-

fects had no bearing on the ultimate outcome.

CONGESTIVE HEART FAILURE

Congestive heart failure contributed 8 deaths in 186 operations, a 4.3 per cent fatality. This compares to a 2 per cent mortality in 314 operations in patients with compensated heart disease. All of these patients in failure were receiving digitalis and or salt free diet and were therefore considered to be compensated before surgery was performed. The degree of myocardial reserve is the most important single factor in determining the mortality and morbidity in patients with heart disease who undergo surgery. With the proper use of oubain and strophanthin and or digitalis, patients can be readied for surgery even in the most urgent circumstances. Congestive heart failure, untreated, adds considerably to the surgical risk. Even when adequately treated, congestive heart failure constitutes a definite hazard to an operation; and it is here, more than anywhere else, that choice of anesthesia and anesthetist is of utmost importance.

MURMURS

Murmurs per se played an insignificant role in the mortality statistics. In 127 patients with organic murmurs there were 4 deaths (3.1 per cent mortality) compared to 10 deaths in 373 operations (2.6 per cent mortality) in patients without organic valvular lesions. Only 1 of these deaths was in rheumatic heart disease with mitral and aortic stenosis. The others were in arteriosclerotic heart disease with aortic stenosis and mitral insufficiency. It is generally conceded that inactive or chronic valvular defects do not alter the mortality rate.

CARDIAC ENLARGEMENT

Cardiac enlargement (Figure 5) not only did not increase the mortality rate but actually was associated with a lower incidence of fatalities (1.6 per cent) than patients with a normal sized heart (4.4 per cent).

ANGINA AND CORONARY THROMBOSIS

Patients with a history of angina are probably considered by many as extremely poor surgical risks; whereas, the mortality rate in 99 operative procedures in this series was only 2 per cent (Figure 5). The occa-

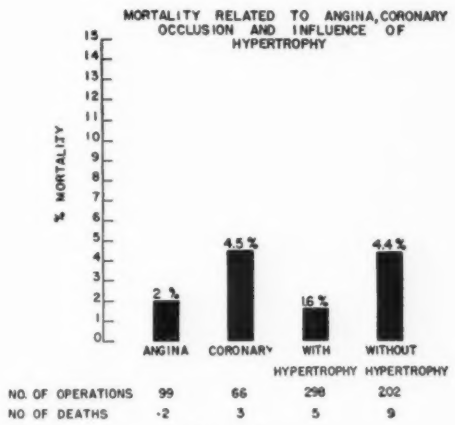

Figure 5

sional viewpoint of prohibiting all except the most urgent emergency surgery in patients with angina cannot be confirmed by this study. It is wise, however, to treat anginal symptoms with great respect and to ignore such pseudocriteria as the electrocardiogram in favor of clinical judgment. Unless a patient is having angina decubitus or status anginosis, this syndrome should not in itself contraindicate necessary surgery, whether emergency or elective. Patients who have had coronary occlusion with infarction can also tolerate surgery fairly well. There were only 3 deaths in 66 operations, a mortality of 4.5 per cent. Common sense dictates waiting three to four months or longer after acute coronary occlusion if at all possible before attempting elective surgery. La Due *et al* report a mortality of 5.8 per cent in 58 patients with healed myocardial infarcts undergoing major surgical procedures. They concluded that a healed myocardial infarction is not a contraindication to surgery and that the surgery had no adverse effect on the heart disease.

MORTALITY RELATED TO FUNCTIONAL CAPACITY

All patients were classified as to their functional status (Figure 6) in accordance with the criteria set forth by the American Heart Association, namely: Class I—Patients with a cardiac disorder without limitation of physical activity; ordinary physical activity causes no discomfort. Class II —Patients with a cardiac disorder with

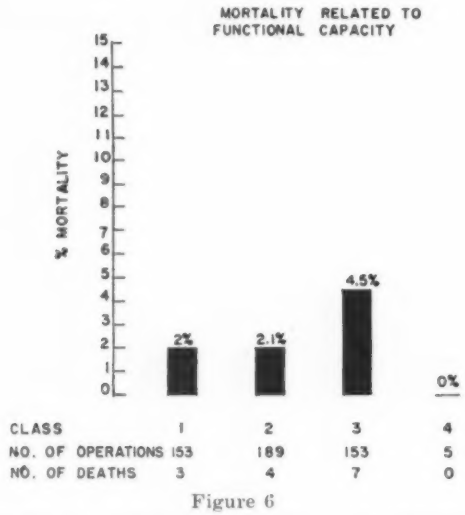

Figure 6

slight to moderate limitation of physical activity; ordinary physical activity causes discomfort. Class III—Patients with a cardiac disorder with moderate to great limitation of physical activity; less than ordinary physical activity causes discomfort. Class IV—Patients with a cardiac disorder unable to carry on any physical activity without discomfort.

Generally speaking, the mortality was directly proportional to the functional class, being 4.5 per cent in the Class III category as compared to 2 per cent in Class I.

MORTALITY RELATED TO TYPE AND DURATION OF OPERATION

The 500 operations which formed the basis for this study consisted of numerous types of surgical procedures (Figure 7).

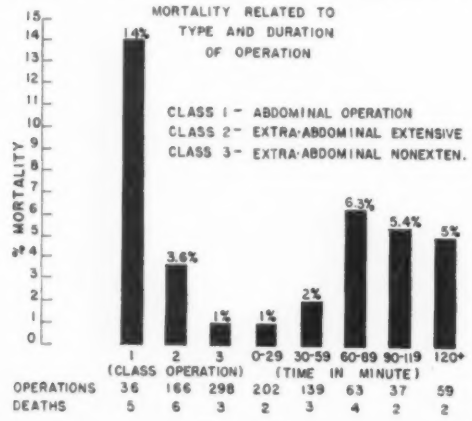

Figure 7

In order to avoid the misleading terms "major" and "minor" surgery, these operations were divided into three groups—Group I comprised all intra-abdominal procedures. Group II consisted of all extra-abdominal extensive procedures and Group III included all extra-abdominal nonextensive operations. Group I, or the abdominal group, had an unusually high mortality figure (14 per cent) but consisted of the smallest number of cases (36). Group II operations resulted in a 3.6 per cent mortality and Group III, 1.0 per cent. In a further analysis of these procedures, the length or duration of the operation bore a definite relationship to the death rate, there being a progressive increase up to sixty to eighty-nine minutes and a leveling off after one and one-half hours. It would seem, therefore, from these figures that patients requiring abdominal surgery are subjected to a somewhat greater risk than those undergoing nonabdominal procedures whether the operation is of a so-called "major" or "minor" nature, and that the risk is greater in operations lasting over sixty minutes.

MORTALITY RELATED TO ANESTHETIC AGENT

The anesthetic agent has a definite bearing on the ultimate outcome, although no deaths in this series of cases could be directly attributed to the type of anesthetic agent employed (Figure 8). In this series

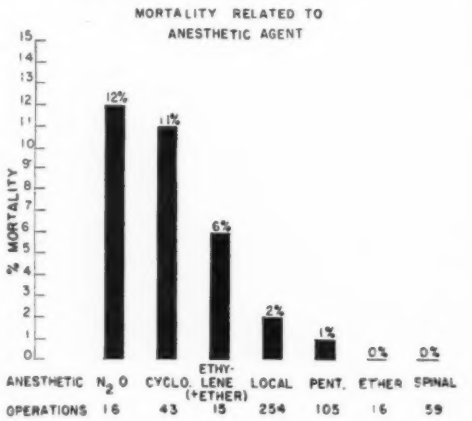

Figure 8

of cases the human factor was rather constant, inasmuch as the same anesthestist or group of anesthestists administered or

supervised the administration of anesthesia in almost all of these surgical procedures. The different agents are listed in the accompanying table according to their relative safety, with spinal, ether, pentothal, and local heading the list. These figures are based on the primary anesthetic agent used although in some instances a combination of agents was employed. Spinal is usually condemned for its hypotensive effect and consequent danger of peripheral vascular collapse and diminished coronary blood flow, although Master and Jaffe actually recommend spinal anesthesia for its peripheral vasodilatory effect. We merely point out that in 59 operations where patients received spinal anesthesia, the mortality was nil. There were no deaths with ether when used alone in 16 cases and only 1 death in 105 patients who were given pentothal. Ethylene alone or combined with ether resulted in 1 death out of 15 cases, resulting in a mortality of 6 per cent. Cyclopropane contributed 5 deaths in 43 operations for a mortality of 11 per cent. Nitrous oxide also had a high fatality with 2 of 16 procedures proving fatal, a mortality of 12 per cent.

COMPLICATIONS

The complications and causes of death are listed in Figure 9. Shock, pneumonia, and congestive heart failure comprised over 60 per cent of these complications, the remainder being pulmonary embolism, atelectasis, pleural effusion, congestive heart failure, coronary occlusion, thrombophlebitis, acute renal failure, and auricular fibrillation.

CONCLUSIONS

1. Generally speaking, patients with heart disease withstand surgery and anesthesia with a minimum of additional risk.

2. Proper evaluation and management of the cardiac patient is essential.

3. Factors increasing the mortality in this series were the presence of arteriosclerotic heart disease, congestive failure, auricular fibrillation, coronary thrombosis, functional capacity, type of surgical procedure, duration of operation, and the anesthetic agent.

4. No appreciable increase in mortality was observed in this series in the presence of syphilitic, rheumatic, and hypertensive heart disease. Race, conduction defects, murmurs, cardiac enlargement, and angina likewise resulted in no increase in mortality rate.

5. Serious cardiac complications, e.g., acute coronary occlusion and cardiac arrest are rare as a result of surgery.

COMPLICATIONS

SHOCK	14
PNEUMONIA	7
HEART FAILURE	6
PULMONARY EMBOLISM	3
ATELECTASIS	2
THROMBOPHLEBITIS	2
C V A	1
CARDIAC ARREST	1
APNEA	1
CORONARY OCCLUSION	1
PLEURAL EFFUSION	1
AUR FIBRILLATION	1
ACUTE RENAL FAILURE	1

Figure 9

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PERICARDIAL EFFUSION FOLLOWING VIRUS PNEUMONIA

A CASE REPORT

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The occurrence of pericardial effusion as a complication of atypical pneumonia is uncommon. In 1938, Willius[4] stated that 68 per cent of pericardial effusions were due to acute infectious types of diseases and occurred secondary to other intrathoracic infections. In 1941, Willius[5] reported a case of acute serofibrinous pericarditis following acute pharyngitis. Nathan and Dathe[1] reported on pericarditis with effusion following apparently trivial upper respiratory infections. Wolff[6] has reported 3 cases (cases 2, 4, and 5) in which primary atypical pneumonia was complicated by acute pericarditis without effusion. In their series of cases of acute pericarditis, Nay and Boyer[2] reported 1 case (case number 42) in which pericardial effusion occurred in the course of atypical pneumonia. Two cases (numbers 1 and 2) of acute pericarditis described by Taubenhaus and Brams[3] may have had virus pneumonitis concomitantly which was not recognized. Probably many more cases of "idiopathic" pericarditis, with or without effusion, have occurred as a result of atypical pneumonitis which was not recognized.

Since the advent of the antibiotics, the treatment of pneumonia has resulted in more cures and less of the complications such as empyema, pericarditis, and pericardial effusion, with their attendant high mortality. With the discovery of the newer broad spectrum antibiotics and their beneficial effect on the recently recognized virus pneumonia, the treatment of chest infections has been greatly simplified and this may result in some degree of complacency on the part of the physician. The diagnosis of virus pneumonia is being made more frequently and one should be aware of the possible complications. The following case is presented with this in view.

CASE REPORT

Mrs. W. S., a 35-year-old white female, was first seen on October 4, 1951. At that time she was complaining of a dry hacking cough and pain in the chest posteriorly on deep inspiration. There were no other symptoms. Past history was noncontributory and there was no history of tuberculosis, rheumatic fever, malaria, or typhoid. Her temperature was 98.8 F°; blood pressure was 120 mm. Hg. systolic and 75 mm. Hg. diastolic; pulse rate was 76/min. regular and full; respiratory rate was 20/min. On examination she was found to have some fine rales in the left chest posteriorly. The rest of the physical examination was negative. A diagnosis of virus pneumonia was made and she was treated with chloromycetin, 500 mgm. stat and then 250 mgm. every four hours thereafter.

She responded very well to treatment and was so much improved that after three days she required no further treatment.

On the evening of October 10, 1951, her husband returned home to find her sitting up in bed struggling for breath and unable to speak. On examination in the home at 11:00 P.M., the patient was sitting in the bed upright, unable to speak, orthopneic, dyspneic, apprehensive and apparently unaware of her surroundings. Her face was greyish-blue, the finger and toenails were blue, the skin was cool, the neck veins were engorged. Temperature was 99 F°. Blood pressure was 90 mm. Hg. systolic and 40 mm. Hg. diastolic; pulse was 130/min. regular, rapid and faint, respiratory rate was 30/min. short and labored.

The pertinent findings on physical examination were: Marked facial weakness bilaterally, paralysis of the glossopharyngeal nerves, inability to protrude the tongue, inability to phonate. There was marked generalized weakness of the limbs, there was no hypotonia or atrophy, the sensory system

*Former resident in Pathology, Touro Infirmary, New Orleans, Louisiana.

could not be examined; reflexes were normal bilaterally. The chest was clear. The apical impulse could not be felt, the heart was apparently not enlarged on percussion. The heart sounds were faint, distant, rapid and regular at 130/minute; there were no murmurs. The liver edge was palpable on inspiration and tender.

She was immediately given coramine and transferred to a local hospital where she was placed in an oxygen tent and given intravenous fluids. Chest x-ray on admission (Figure 1)* showed an area

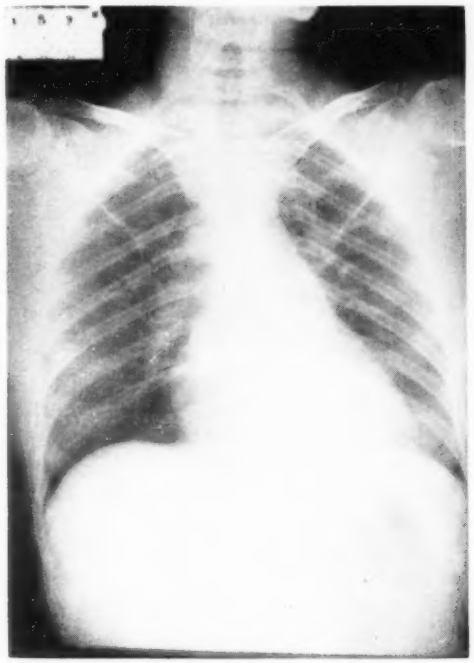

Fig. 1.

of increased density in the left lower chest which was in keeping with her pneumonitis. The cardiac shadow was markedly enlarged; the cardiodiaphragmatic spaces were almost obliterated; the edges of the cardiac shadow were rounded and enlarged to the right and the left. This picture was taken with a portable machine at the bedside. Flouroscopic examination was not done because of the patient's poor condition.

On re-examination the area of cardiac dullness was enlarged to the right and left; the area of dullness was larger in the second and third interspaces bilaterally with the patient flat in bed; with the patient erect, the area of dullness in these spaces was smaller but was increased in the right and left fourth and fifth spaces-shifting cardiac dullness.

*The author wishes to express his grateful appreciation to Dr. Louis Raider, Radiologist, Mobile, Alabama, for his invaluable assistance.

These further signs on examination, along with the clinical and x-ray findings were taken as evidence of pericardial effusion rather than acute dilatation of the heart.

Laboratory data: Red blood cell count—4.4 million; Hemoglobin—86% (12.2 gms.); White blood cell count 11,400; Differential—neutrophiles 48%; lymphocytes 52%. Urinalysis—specific gravity—1.026; negative for albumin; negative for sugar; microscopic negative. Stool—negative for ova and parasites. Serology—(Kahn reaction)—negative.

Pericardiocentesis was considered but since she was not becoming worse and was apparently comfortable in the oxygen tent, the hazards of the procedure were thought to outweigh the benefits she might derive, and therefore, aspiration was not done.

Since the original infection was of virus origin and had caused a pericardial effusion, it was thought she might have a pericarditis and a pericardial effusion due to the same virus. Therefore treatment was begun on October 12, 1951, with chloromycetin, 500 mgm. every four hours. Later that same day she regained her speech, was able to protrude her tongue, and the weakness of the facial and glossopharyngeal nerves was decreased. However, she complained of pain and tightness in the centre of her chest which were increased by movement, respiration, and coughing—substantiating the presumptive diagnosis of pericarditis.

The next day she seemed further improved, having no chest complaints, and was very comfortable in the oxygen tent. She was allowed out of the tent for one half hour twice that day; this resulted in dsypnea and slight cyanosis; therefore, she was kept in continuous oxygen.

Despite these improvements following chloromycetin therapy, the physical findings of an increased area of cardiac dullness and enlarged liver remained unchanged. On October 16, the chloromycetin was discontinued since it was an economic strain on the patient. She was then fully digitalized with digitoxin to relieve the right-sided mechanical congestive failure due to tamponade. Two days later the area of cardiac dullness was apparently of normal size on percussion and the liver was not enlarged.

By then the facial and glossopharyngeal weakness had completely disappeared; her pulse and respiratory rates had returned to normal; she could tolerate being out of oxygen tent and she was allowed to be out of it for increasing periods of time.

A portable chest plate on October 19, 1951, (Figure 2) showed the cardiac shadow to be decreased in size with less convexity along each of its borders, apparently the result of absorption of the fluid.

On October 23, digitoxin was discontinued. By then she had no complaints and physical examination was normal. She was discharged on October

Fig. 2.

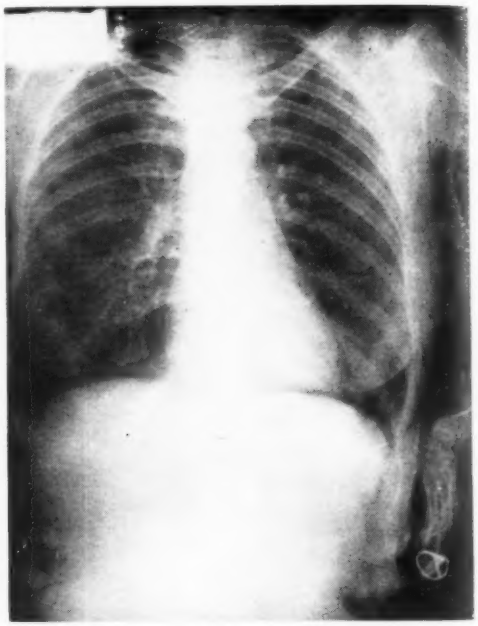

Fig. 3.

25 to convalesce at home and all medication was discontinued.

Throughout the hospital stay her temperature was normal; her blood pressure rose gradually and was 106 mm. Hy. systolic and 80 mm. Hg. diastolic on discharge.

On November 29, 1951, she was seen in the office for a check-up and she had no complaints. She remembered nothing about her recent illness until she awoke in the hospital on October 12. She stated that she remembered hearing her husband say, "The doctor is coming," but she could not see her husband although he was standing at her bedside in their home shortly after the onset of her dyspnea and cyanosis of which she was unaware and did not understand why the doctor was coming. This incident had occurred several minutes before the patient was seen in her home and by then she was unaware of her surroundings and could not speak. Physical examination was negative and she was discharged.

On January 12, 1952, she was seen for other complaints and at that time re-examination showed no findings relative to her illness in October, 1951. Upright chest plate on January 14 showed a normal chest and a normal cardiac shadow. (Fig. 3).

COMMENT

This case is unusual in that very few cases of pericarditis and pericardial effusion have been reported following virus pneumonia. In addition, temporary nerve paralysis as a result of these complications has not been reported in the available literature. The mechanism of the paralysis, as hypothesized by the author, may have occurred as follows: the pericardial effusion caused decreased return of blood from the great veins due to cardiac tamponade, resulting in decreased flow of blood to the great vessels, leading to relative anoxemia, cyanosis, and cerebral anoxia. The cerebral anoxia resulted in aphonia, temporary facial and glossopharyngeal paralysis through temporary damage due to lack of oxygen to the cortical cells or the nerve ganglia. It it interesting that her visual centre lost its function before the auditory centre, and that she has a memory gap for a period of about thirty-six hours.

SUMMARY

A case of pericarditis and pericardial effusion resulting in cerebral anoxia and temporary nerve paralysis is presented as a rare complication of virus pneumonia. It is suggested that some cases of "idiopathic" pericarditis and pericardial effusion are probably complications of virus pneumonitis.

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ADDRESS*

LOUIS J. BRISTOW, D. D.

NEW ORLEANS

I am not a doctor, but for more than forty years I have worked with them, and always with increasing admiration because of their readiness to serve, with or without compensation, in any case of sickness or accident to which they may be called. My field has been in hospital administration, and through the years there has been a mutual understanding of our respective problems, mutual sympathy and close co-operation. In this day of complex and ever multiplying demands upon his profession, the doctor is well-nigh helpless without the hospital, and it is certain the hospital could not function without the cooperation of the doctor; and I have had ample opportunity to learn the doctor will give his best to make the hospital what it ought to be if he is permitted to do so. I gladly pay them the well deserved tribute of my gratitude for their loyalty and helpfulness in giving whatever measure of success may have attended my efforts in the hospital field.

One who knows doctors well cannot but like them and have a tender feeling for them. The very nature of their work appeals to one. It has well been said that "physicians have undoubtedly helped to put a kindlier spirit into humanity." That testimony is true, and if there were no other reason, that alone should give them a secure place in the hearts of their fellow men. But the doctor is not dependent upon that claim to the affectionate regard of men. Other reasons abound. His service to the race has been and is inestimable. The roster of the world's great men is incomplete without the names of many distinguished doctors.

In my judgment doctors have not been given their proper place in history. Long it has been a matter of wonder to me that our schools give such disproportionate attention to the great warriors of the past, while those who have served mankind in the quieter, gentler realms of peace are generally neglected. Every school child early is made familiar with the Alexanders, the Caesars, the Napoleons, the Wellingtons, and the Hitlers of history; men whose way to greatness has been made hideous with the dead and mangled bodies of the slain over which they have marched. The smoke of destruction has darkened their paths and above the acclaim of their victorious hosts is heard the wail of suffering from multitudes of new-made widows and orphans.

I believe our children should be taught something of the work of those whose lives have been devoted to the service of mankind in the gentler arts of healing. I believe school pupils should be made familiar with the names of those who have employed their talents and their energies to heal rather than to hurt. The boys and girls of today should be taught the history of those whose efforts have been not to destroy life, but to preserve it. The doctor:

> "I reckon him greater than any man
> That ever drew sword in war;
> I reckon him nobler than king or khan,
> Braver and better by far."

I assert without fear of successful contradiction that Harvey, who demonstrated the circulation of the blood, was a greater benefactor of the race than Napoleon with all his invasions; that Long, in his discovery of anesthesia, rendered a greater service to the world than Alexander the Great by all his conquests; that Joseph Lister in giving us the method of antiseptic treatment of wounds, did a greater work for humanity than did his illustrious fellow Englishman Oliver Cromwell, in dethroning Charles I; that Professor Koch of Berlin, when he isolated the tuberculosis bacteria, served mankind better than did the German statesman Bismarck.

*Presented at the Seventy-second Annual Meeting of the Louisiana State Medical Society, Shreveport, April 28, 1952.

Why should we acclaim the virtues of war and fail even to mention the marvelous achievements of peace? In reality, doctors have been man's greatest benefactors—greater than inventors, and discoverers, and statesmen, and diplomats, and artists. In their dissecting rooms and laboratories they have fought the battles of peace for the healing of humanity's hurt. In their workshops they have invented instruments and implements for the relief of suffering. They have discovered methods of treatment of sickness and accident whereby men have been blessed immeasurably. All these have been victories of peace; and uet us not forget that it was the Divine Physician who Himself was called the Prince of Peace.

The healing art has made men compassionate in their ministries to the sick. An indelible pattern of human unity has been woven into the fabric of medical life. In a world of ruthless violence and confusion, by their practice doctors constanly have reaffirmd their undiscourageable belief in the ultimate triumph of friendliness amongst men. Theirs is the task to keep alive in the human breast a hope of relief from pain and the cure of disease. Statesmen of the world long have been trying to bring about international unity, which is an end much to be desired. Language and customs have isolated nations into independent units. Only the healing art is definitely internationalized. It is in the realm of physical healing that the best example of international cooperative life is seen. Contributions of American doctors have been many and varied. But also have been the contributions of Russians, Englishmen, Japanese, Germans, Frenchmen, Austrians, and Canadians. I could call names, but you already are more familiar with them than I. These men did not think in terms of national boundaries, but the whole world was their field. They have sought to serve all mankind. The finest products of their minds and of their labors always have been made available to serve without regard to race or color or nationality or creed. Statesmen may labor to achieve unity in political and economic life and their efforts may be futile. But in the realm of healing already it is a fact. The healing of humanity's hurt cannot be hedged in by geographical boundaries, or governed by commercial advantage; and we owe this to doctors.

Of all men, doctors should be religious folk. Many of them are indeed deeply spiritual. The ethics of Judaism and the principles of Christianity are their guiding stars. It is the doctor who witnesses the coming into the world of new life. He stands daily face to face with this miracle, and can but turn his thoughts to the Author of life. It is the doctor who is in daily contact with physical ills, diseased and distorted bodies, who witnesses the miracle of healing; which he can explain only by referring it to the Author of health. It is the doctor who accompanies his patient to the very edge of the grave, who looks on in utter helplessness as the spirit leaves the body—the mystery and miracle of death. Surely his daily experiences are such that he is constantly brought to face the question of another world, of life beyond the grave. If he is a thoughtful man, he must needs think on the deep things of life, even spiritual things. How often does he need the counsel and aid of the Great Physician! And the truly wise doctor seeks His guidance and blessing.

No one knows better the relation of religion and health. He knows the physical effect of spiritual life. He knows that soul and body react upon each other, and that health is largely dependent upon mind, or spirit. It is well known among doctors that there are physical diseases that cannot be understood or cured unless the spiritual factors in the history of the case are taken into account. Hence, the psychiatrist with his splendid contribution to healing in the realm of personality or emotional disorders. I could enlarge upon this thought; but suffice it to say that man's love of his fellowman is well illustrated by the accomplishments of the various departments of medicine in these modern times. Not a few doctors have literally given their lives in devotion to their tasks of research, as you well know. Moreover these research men and

women attribute to God their discoveries respecting disease, their cause, their cure, and their prevention. A well known illustration of this is seen in the reaction of Sir Ronald Ross, who, after long, unremitting toil, found conclusive proof of the causal relation of a certain type of mosquito to malaria. Dr. Ross wrote:

"This day relenting God hath placed within my hand
A wondrous thing: and God be praised: at His command
Seeking His secret deeds with tears and toilsome breath
I find thy cunning seeds, O, million-murdering death.
I know this little thing a myriad men will save;
O Death, where is thy sting, thy victory, O Grave!
Before Thy feet I fall, Lord, who made high my fate,
For in the mighty small is shown the mighty great."

Sir Ronald's "fate" indeed was made high, for his name is inscribed among the illustrious ones who have served humanity well through self-sacrificing labor. He is for all time famous because he forgot himself "with tears and toiling breath"; and the results of his work have saved a "myriad" from the "million-murdering" insect's bite. We have it upon Supreme Authority that "Whosoever shall seek to save his life shall lose it; and whosoever shall lose his life shall preserve it." Sir Ronald's name will not perish.

Through many centuries the priest or preacher and the healer was one and the same person. The minister of religion was also the minister of medicine. In early American history the minister often practiced medicine, and the names of some illustrious men adorn the roster of such practitioners. Advancing complexities of civilization, and increasing knowledge had a tendency to cause men to become specialists. Some were better qualified to preach while others were more interested in ministering to the sick. Indeed the Divine way is for men to specialize. The inspired Apostle tells us:

"There are diversities of gifts, but the same Spirit . . . For to one is given by the Spirit the word of wisdom, to another the word of knowledge by the same Spirit . . . to another gifts of of healing by the same Spirit . . . dividing to every man severally as He will" (I. Cor. 12:8-11).

But certain men in high places are trying to limit the freedom and control the activities of doctors in this country, under the guise of public benevolence. Call it planned economy, or the Welfare State, or by whatever term you may, socialized medicine is an attempt to reverse and to repudiate the time-honored, time-tested philanthropic service of the medical profession. It is a contradiction of the Christian concept upon which this nation was founded and under which it has reached a place of world leadership. The spirit which has inspired and made possible the amazing achievements of the medical profession is the right of the individual to make his own decisions, to accept the responsibility for them, and the consequence of them.

Will the federalization of medical service improve it? Heretofore the initiative for such service has been found in the loftiest of all human impulses—that of the personal will to serve one's fellows. Within the last week we have seen in the public press two typical illustrations of that fact. Dr. W. L. Ervin of Inverness, Miss., was a man who practiced in a rural community for half a century, never declining to respond to a call, who never sent a bill to a patient for his services. As he lay upon his deathbed and the story of his long, self sacrificing life was published in the daily press, letters from every State in the Union were received testifying to his humanitarianism in serving the writers in their time of need. The old man died poor in this world's goods, but rich in the affections of those whom he had loved and served. The other case was that of Dr. John Wesley Simpson of Parrish, Ala., another country doctor, who observed his eightieth birthday by cancelling every account due to him by his patients—a sum aggregating nearly forty thousand dollars. In the light of such instances, I repeat my question: Will the federalization of medical service improve it? Federalization cannot supply the motive necessary to give efficient service. The

thought that the Government through mechanical perfection can ever take the place of service prompted by an enlightened Christian humanitarianism is a glittering illusion held out by self seeking individuals or misguided politicians. The decision of the Supreme Court of the United States, written by Justice Brewer, handed down nearly a generation ago, that this is a Christian nation, still stands. We are basically Christian. Evidence of this is seen in the reaction of press and pulpit and the man in the street to the recent exposures of corruption in the Government at Washington, the income tax scandals, to the cheating at West Point, the scandals among college athletic teams, and wrongdoing in other places. That reaction affirms that ours *is* a religious people; and I assert that we are endowed of God with individual dignity and independence and responsibility. The concept of an all-powerful Welfare State is pagan, and contravenes the Christian idea of life. Doctors and hospitals through the Blue Shield and the Blue Cross systems are offering a better medical plan—a plan which promotes individual personal dignity and responsibility.

In the preamble to the constitution of this country, the founding fathers declared that document was ordained and established to "secure the blessings of liberty to ourselves and our posterity." We cherish with high veneration and grateful appreciation the memory of our fathers. Both the ties of nature and the dictates of policy demand this; but we are today witnessing a vandal spirit of innovation and overthrow of long established and successful customs. Eternal vigilance is the price of liberty, and we should not relax our opposition to the efforts which are being made to socialize your profession. The proponents of such an idea may be quiet now, but they have not surrendered or given up hope.

NEW ORLEANS
Medical and Surgical Journal

Established 1844

Published by the Louisiana State Medical Society under the jurisdiction of the following named Journal Committee:

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SUBSCRIPTION TERMS: $4.00 per year in advance, postage paid, for the United States; $4.50 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Ave., New Orleans, La.

THE JOURNAL *does not hold itself responsible for statements made by any contributor.*

1952 DIABETES DETECTION DRIVE

The American Diabetes Association will launch its fifth nationwide Diabetes Detection Drive during the week of November 16-22. With the assistance given by the Association, utilizing the facilities of organized medicine, the opportunity will be presented to the individual physician to do distinct service to the community and to the individuals in his practice.

About 30 State and nearly 700 County medical societies throughout the nation will participate in the program this year. The drive is a nonfund-raising, educational, and case-finding program. It is directed exclusively through the medical profession. The membership in the American Diabetes Association is limited to physicians. Detailed surveys in certain localities, and the experience of the past four years have indicated that there are probably one million diabetics under treatment in this country, and yet another million who are suffering from diabetes, undetected, and therefore, untreated.

The control of diabetes can only begin when it is detected. Control means better health for the patient, and, very often, for the family group. The objectives of the American Diabetes Association are:

To find the greatest number possible of yet undiscovered diabetics.

To assist diabetics in their efforts to lead normal lives.

To improve the treatment of diabetes.

To bring the newest information about the disease to all interested physicians.

To encourage and support research on diabetes.

To promote public knowledge about diabetes and understanding of the individual diabetic's problem.

The efforts of the Association are focused on the Detection Drive. Each physician can help.

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WHAT CONSTITUTES ADEQUATE CONTROL OF DIABETES

The physician who has diabetics under his care must inquire what constitutes adequate control of the condition. The Diabetes Guide Book of the American Diabetes Association states that from the physiological point of view the objective of treatment is to keep the urine sugar-free and the blood sugar normal. Further objectives from the physical standpoint are:

To relieve symptoms.

To restore and maintain normal vigor and optimal body weight.

Also from a personal viewpoint of the patient:

To enable him to share as completely

as possible in the normal enjoyment of living.

There seems to be no disagreement on any of these objectives except the first. The opinion varies on this point with various grades of procedure and regulation. The conservative writers state that they strive for normoglycemic control and aglycosuria. The liberals advocate a free diet and disregard all but symptoms, acidosis, and weight status. Between these two points of view is a great gulf fixed, and into this gulf has stepped the average diabetic with a syringe full of insulin and a desire to eat a so-called normal diet.

The survey by Beaser[1] from a questionnaire sent to 300 physicians of the American Diabetes Association demonstrates the diversity of opinion. Two hundred and twenty-five replies were received from these representative members of the association. The aim of therapy was normoglycemia among 70 per cent, aglycosuria among 23 per cent, and glycosuria permitted by 7 per cent. There seems to be reasonable consensus of opinion that the majority who advocate normoglycemia recognize special situations in which reluctant compromise is necessary from a practical point of view. There seems to be fair agreement that the diabetic should be slightly under his theoretical normal weight. It appears that a minimal protein need of 1 gram per kilogram is necessary and some advocate 1.5 grams. The allowance of fat is to be less than 120 grams. The allotment of carbohydrate is varied according to the necessities indicated by the changes in weight. Most diabetics are felt to require only relative constancy of the daily dietary intake within plus or minus 10 to 15 per cent.

The desirability of maintaining as precise control as practical applications of diabetic principles will permit is shown by the following survey: A group of 247 patients whose diabetes had started between the ages of 18 months and 30 years, and who had had the disease for from ten to thirty-four years, was studied by Wilson, Root, and Marble.[2] Particular effort was made to study the development of degenerative lesions in the kidneys, retina, and blood vessels. These were studied by radiograms of the abdomen and legs, renal function tests, and funduscopic examinations. No patient with good control of diabetes had advanced calcification or retinitis. All but 5 of the patients with moderate to severe retinal lesions had had fair to poor control. Of the 62 patients who had diabetic nephropathy, all had maintained poor to fair control. Control was considered to be more significant than duration or severity of diabetes in preventing vascular lesions. Patients who had lived on a measured diet with constant use of insulin and careful control of glycosuria were regarded as having had good control.

The prevalent opinion seems to be that blood sugar examinations are the only adequate guide for the control of abnormal hyperglycemia.

John[3] reported in a study of 6000 diabetics that glycosuria occurred in 6.3 per cent of these diabetics at a time when the blood sugar level was 120 milligrams per cent or less, and that the renal threshold may be found anywhere from 50 to 600 milligrams per cent of blood sugar. In 8.2 per cent the renal threshold was above 200 milligrams per cent. Accordingly, glycosuria can only be used as a guide to the existence of an abnormal hyperglycemia after the renal threshold has been determined. John[4] uses a restrictive qualitative diet of 2000 to 2500 calories and allows hyperglycemia two hours after each meal or about 25 per cent of the twenty-four hours, which he considers occurs in a normal and constitutes good control. Duncan[5] stresses that

[1]Beaser, Samuel B.: New England J. Med. 244: 714 (May 10) 1951.

[2]Wilson, J. L., Root, H. F., and Marble, A.: Am. J. M. Sc. 221:479, 1951.

[3]John, H. J.: Ann. Int. Med. 33:925, 1950.

[4]John, H. J.: Ann. Int. Med. 35:1318, 1951.

[5]Duncan, G. G.: Diabetes Mellitus; Principles and Treatment, W. B. Saunders Co., Philadelphia, 1951, pp. 112-116.

there should be uniformity; that is, the food values for breakfast, lunch, and dinner should be the same day after day.

Adequate control of the diabetic thus appears to be obtained by those who aim at normoglycemia and depend upon blood sugars for the estimation of insulin dosage and the routine regulation of the diet.

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ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

SEVENTH ANNUAL NATIONAL AND LOCAL ESSAY CONTEST

We should all show greater interest and encourage more pupils in our schools to participate in the 1953 A.A.P.S. essay contest sponsored by county and state medical societies, auxiliaries and schools. The subject, as you know, is, "Why the Private Practice of Medicine Furnishes this Country with the Finest Medical Care." We should be ever ready and willing to enlighten our students in this wonderful educational undertaking, which is more or less continuous and endless as we are presented each year with a new crop of high school students who are eager to secure knowledge and should be informed on this most important question of our time: Freedom versus Socialism.

It is very essential that these young people be properly advised against the pitfalls of socialized medicine and the many advantages of the free enterprise way of life to be enjoyed in the private practice of medicine as we have it in this country at the present time. You must realize that the high school girls and boys of today are the voters of tomorrow, and unless we intelligently educate them against this present day trend of socialism and correct the misconception of some of this younger group who might believe in or favor socialized medicine, we might wake up some bright morning to find that these grown-up people have voted us into socialized medicine.

We find that this essay contest has been our greatest asset in educating these young students against the evils of socialism. It also, at the same time, helps their parents to think in the right direction.

There are grand local and national prizes offered the winners of this contest, and from an educational standpoint, it merits the support and approval of our state and parish medical societies.

Why not talk to your Parish Superintendent of Education or to the principals of the schools, eliciting their cooperation in getting more of these essay contests started in your community? This would be a wonderful public relations program for the good of all. For your information, Dr. Edwin L. Zander, our immediate Past President, is National Chairman of the 1953 Essay Contest Committee sponsored by the Association of American Physicians and Surgeons with the cooperation of the American Medical Association, state and parish medical societies and the woman's auxiliaries of these respective organizations.

Why not start a crusade to enlist more of our people in becoming interested in this most worthy undertaking?

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VOTE

Your vote is extremely important as is shown in the following message brought to our attention by the National Association of Manufacturers.

All efforts should be made to convince each voter of our state of the great importance of his or her vote in the elections to be held at an early date. You have only a few days to perform this most urgent task.

Let us not fail to show a high percentage of votes cast by our physicians in the coming elections.

It is our opinion that a greater number of our doctors should take a more active part in local, state and national politics for the good of the profession. However, it seems that a few of our members evidently think that we should sit idly by and watch our house burn down.

One Vote never seems important—*before* an election.

But on the day *after*—on November 5, this year—someone, somewhere will be saying: "If we'd only had *one more vote!*"

It may happen in a "little" election—for township trustee, or a county officer, or a rural school board member—or for the Presidency of the United States.

It *has* happened, many times in our short history.

One vote cast by a voter who left his sick bed to go to the polls gave victory to an Indiana Congressman . . .

One vote cast by this same Congressman, in 1876, made Rutherford B. Hayes President of the United States . . .

One vote cast by a miller in DeKalb County, Indiana, in the 1840's, elected a certain man to the state legislature . . .

One vote cast by this same legislator, sent Edward A. Hannegan to the United States Senate . . .

One vote cast by the same Edward Hannegan, admitted Texas to Statehood—for which U. S. and Mexico *went to war* . . .

One vote admitted California, Idaho, Washington and Oregon to the Union . . .

One vote in the U. S. Senate denied this statehood to Alaska . . .

One vote more in each California precinct in 1916 would have defeated Woodrow Wilson, and given victory to Charles Evans Hughes . . .

One vote more in each of Ohio's and California's precincts would have tied the electoral race between Dewey and Truman in 1948 . . .

One vote in November could change world history . . .

Will it be *yours?*

"Worth Thinking About"

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PHYSICIANS URGED TO MAKE RESERVATIONS IMMEDIATELY FOR A.M.A. MEETING

Physicians who expect to attend the clinical sessions of the American Medical Association to be held in Denver, Tuesday-Friday, December 2-5, should make their reservations at an early date. You will find in the A.M.A. Journal hotel reservation and advance registration forms for your convenience.

Meetings of some special groups will be held prior to the regular meeting, which will open Tuesday morning, December 2, in Denver's newly enlarged Municipal Auditorium, housing the scientific exhibits, medical motion pictures and technical exhibits.

Registration will also take place in the Auditorium.

Interesting colored television with general lectures and clinical presentations will be on the same floor with the exhibits.

These meetings are extremely instructive and cover a wide range of approximately 200 lectures on vital subjects of the day in all branches of medicine.

Due to the setup of restaurant facilities in the Auditorium you will be able to spend an entire day, taking advantage of the many splendid programs prepared for you.

Social functions and entertainment programs have been arranged for your evening enjoyment.

Will expect you in Denver on above dates.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

In order to assist specialty groups in selection of dates for meetings and for information of the doctors of the state, information concerning scheduled meetings will be carried in this section of the Journal. Officers of specialty groups are therefore requested to furnish information in this regard.

FELLOWSHIPS GIVEN BY NATIONAL FOUNDATION FOR INFANTILE PARALYSIS

The National Foundation for Infantile Paralysis announces the availability of a limited number of additional postdoctoral fellowships to candidates whose interests are research and teaching in medicine and the related biological and physical sciences. The purpose of these National Foundation fellowships is to increase the number of professional workers qualified to give leadership in the solution of basic and clinical research problems of poliomyelitis and other crippling diseases.

The fellowships cover a period of from one to five years. Stipends to Fellows range from $3,600 to $7,000 a year, with marital and dependency status considered in determining individual awards. Institutions which accept Fellows receive additional compensation for expenses incurred in relation to their training programs.

Complete information concerning qualifications and applications may be obtained from: Division of Professional Education, The National Foundation for Infantile Paralysis, 120 Broadway, New York 5, New York.

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THE INTERNATIONAL ACADEMY OF PROCTOLOGY 1952 AWARD CONTEST

The International Academy of Proctology takes pleasure in announcing its Annual Cash Prize and Certificate of Merit Award Contest for 1952-1953. The best unpublished contribution on Proctology or allied subjects will be awarded $100.00 and a Certificate of Merit. Certificates will be awarded also to physicians whose entries are deemed of unusual merit.

This competition is open to all physicians in all countries, whether or not affiliated with the International Academy of Proctology. The winning contributions will be selected by a board of impartial judges, and all decisions are final.

The formal award of the First Prize, and a presentation of other Certificates, will be made at the Annual Convention Dinner Dance of the International Academy of Proctology in May of 1953.

The International Academy of Proctology reserves the exclusive right to publish all contributions in its official publication, "The American Journal of Proctology and Gastroenterology."

All entries are limited to 5,000 words, must be typewritten in English, and submitted in five copies. All entries must be received no later than the first day of April, 1953. Entries should be addressed to the International Academy of Proctology, 43-55 Kissena Blvd., Flushing 55, New York.

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VA COURSE IN PSYCHIATRY AND NEUROLOGY

The Veterans Administration is instituting a four-month intensive training course in psychiatry and neurology to fit the needs of physicians without such previous training who are assigned to duty in 22 predominantly psychiatric hospitals. Physicians who have been engaged in general practice may request this training upon applying for a position at one of these hospitals.

The course will be held at the VA Hospitals in Coatesville, Pennsylvania; Palo Alto, California; and a joint Downey-Hines, Illinois, program near Chicago, Illinois. Physicians will be employed at salaries commensurate with their training and experience (salary range: $5,500 to $11,800 per annum) and assigned to the course with travel and per diem for the four-month period.

Information and applications may be obtained from your nearest VA Hospital or Regional Office, or by writing to the Chief Medical Director, Veterans Administration Central Office, Washington 25, D. C.

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TELLS DANGERS TO 25,000,000 OVERWEIGHT PERSONS IN U. S.

Obesity is America's number one public health problem.

Approximately 25,000,000 persons in the United

States are overweight to some extent, and at least 5,000,000 adults are seriously obese, according to Louis I. Dublin, Ph.D., New York, statistician for the Metropolitan Life Insurance Company. The problem of overweight affects more people than any other impairment or disease, and is at the root of the high prevalence of the degenerative diseases in this country, he stated.

"Because overweight predisposes these large numbers of people to heart disease, diabetes, liver disorders, and other serious complications, it cannot be ignored by the general public or the medical profession," Dr. Dublin wrote in the current Today's Health, published by the American Medical Association. "The solution of this problem is vitally important for further progress in the health of the nation."

REPORTS NEW METHOD OF TREATING BELL'S PALSY

A new, rapid way of treating Bell's palsy, by local anesthetization with procaine hydrochloride of a small group of sympathetic nerve cells in the neck, was reported in the September 6 Journal of the American Medical Association.

Bell's palsy, a usually innocuous but psychologically distressing disease, is the distortion of the face as a result of paralysis of the muscles on one side of the face. The cause of most cases of the disease is unknown, and treatment has been generally unsuccessful. Although recovery is usually spontaneous, it is long delayed in many instances, causing psychological and economical difficulties.

BOOK REVIEWS

Surgical Treatment of the Motor-Skeletal System; edited by F. W. Bancroft and H. C. Marble, 2 vols., illus. Philadelphia. J. B. Lippincott & Co., ed. 2, 1951. Price $24.00.

This 2nd edition has been edited by Henry Marble who replaced Dr. Clay Ray Murray. There are certain notable general improvements in the second edition. The inclusion of a chapter on bone healing by Marshall Urist, and the revision of certain other chapters notably that on internal derangements of the knee and the chapter on sprains are distinct improvements. However, it is impossible in a book of 1303 pages with 46 contributors dealing with a total of 44 varied sections to maintain continuity of thought or excellency in preparation throughout the book.

It will continue to serve as a useful reference book for those who occasionally must treat orthopedic lesions. It is the reviewer's opinion that although the revision of the text and material have been extensive much material remains which seems to warrant more thorough and complete revision.

JACK WICKSTROM, M. D.

Let's Cook It Right; by Adelle Davis. Harcourt, Brace and Company, New York, 1951. pp. 626. Price $3.00.

Here is a book to excite the reviewer's taste buds, at the same time upsetting some old ideas and throwing new light upon the preparation of food. Although tremendous strides have been made in the science of nutrition, the author feels that the application of this knowledge "has lagged decades behind the progress made in nutritional research." She states that the main purpose of her book, written after thirty years' cooking experience, is "to help build health through applied nutrition which, if successfully applied, makes good cooking a necessity". Loss or destruction of health-building proteins, vitamins, and mineral, results from lack of knowledge of the fundamental of nutrition and she hopes to rectify this condition by presenting "scientific procedures designed to save maximum nutritive value". Tried and recommended basic recipes and their variations are given with thrift and economy stressed. The book, primarily for the housewife, is a readable and enjoyable guide to good cooking and how to avoid "culinary crimes".

Foods that have the greatest concentration of vitamins, minerals and proteins are frequently those which Americans rarely eat, and she points out that "dislike for health-building foods is undoubtedly the greatest single cause of sickness".

MARY LOUISE MARSHALL

PUBLICATIONS RECEIVED

Lange Medical Publications, Los Altos, Calif.: Correlative Neuroanatomy and Functional Neurology, by Joseph J. McDonald, M. D., and Joseph G. Chusid, M. D. (6th Edit.).

The Metropolitan Life Insurance Co., N. Y.: A 40 Year Campaign Against Tuberculosis.

The C. V. Mosby Company, St. Louis: Synopsis of Pathology, by W. A. D. Anderson, M. D., (3rd Edit.).

Charles C. Thomas, Publisher, Springfield, Ill.: Infrared Photography in Medicine, by Leo C. Massopust, Sr.; This is your World, by Harry A. Wilmer, M. D., Ph. D.; Physiological Bases of Gynecology and Obstetrics, by S. R. M. Reynolds, M. A., Ph. D., D. Sc.; Kitchen Strategy, the Family Angle on Nutrition, by Leona M. Bayer, M. D., and Edith Green; The Moral Theory of Behavior, a New Answer to the Enigma of Mental Illness, by Frank R. Barta, M. D.; Finality and Form, by Warren S. McCulloch, M. D.; Neurosurgery in General Practice, by Adrien ver Brugghen, M B.

The Viking Press, Inc., New York: Brain Surgeon, the Autobiography of Dr. William Sharpe.